California MEDICINE

MARCH 1959

OFFICIAL JOURNAL
OF THE CALIFORNIA MEDICAL ASSOCIATION

OLUME 90 NUMBER 3

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Volume 90 MARCH 1959 Number 3

Anticoagulant Drug Treatment of Coronary Artery Disease

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• **Anticoagulant therapy of arteriosclerotic heart disease may prove to be most valuable when applied on a long-term basis for prevention of recurrent myocardial infarction. While its prophylactic value in impending infarction has not been established, at least the accepted treatment for the acute stage is already begun if an anticoagulant has been administered before an inevitable infarction occurs.**

The chief value of the anticoagulant, though, seems to lie in preventing cardiac mural thrombosis and extracardiac thromboembolism. It is by this effect, apparently, that mortality has been reduced by 50 per cent among survivors of myocardial infarction who receive continuous dicoumarin therapy.

While the danger of hemorrhage is still present, it is being steadily reduced by increasing skill in the management of anticoagulant therapy, and for a long time the risk has been far outweighed by the reduction in coronary occlusion.

Physicians have a duty to learn the use of anticoagulant therapy, obtain the facilities necessary for it, and apply it to patients who are able and willing to cooperate in prolonging their useful lives.

ARTERIOSCLEROTIC HEART DISEASE causes over 400,000 deaths annually in the United States,[27] and most of these deaths are due to thrombotic coronary artery occlusion. The ideal treatment is largely a problem of preventive medicine. Hand in hand with measures for the prevention of the primary atherosclerosis must go those for the prevention of secondary thromboembolic complications. The question of possible salvage from antithrombotic drug therapy for prevention of death in myocardial infarction is highly debatable. In 1954 the Committee on Anticoagulants of the American Heart Association concluded from a study of 1,031 cases that the death rate could be reduced by one-third.[28] There are those, however, who still reject the drugs outright; others who would use them only in acute infarction for what they consider "bad risk" cases; and there is the same committee's opinion that anticoagulants should be given prophylactically for three to four weeks in all cases unless contraindicated by a risk of severe bleeding. Still more dubious is the status of long-term anticoagulant drug therapy for preventing a recurrence of myocardial infarction in survivors of the first attack. Some physicians never use long-term therapy, others wait for a second attack or other signs of thromboembolism, and others would use it in every case in which it is not contraindicated. These different interpretations arise from clinical and pathologic statistical studies that are necessarily imperfect. The chances for error in the random selection of patients, and for error and bias in observation, are too great for a perfectly controlled study. The only statistically

Presented as part of a Symposium on Treatment of Intravascular Clotting at the American Heart Association meeting at San Francisco, October 24 to 26, 1958.

Submitted January 16, 1959.

perfect study of anticoagulant therapy would be by the double-blind placebo technique, and such a study is impracticable, especially in the evaluation of long-term therapy.[16]

Despite certain unavoidable technical flaws, the committee's study of 1,031 cases was the most comprehensive possible. Other reports which have disagreed are based on series that were either numerically too small or inadequately controlled. Shall we act on evidence which is only presumptive and not conclusive? Many crucial clinical decisions are based on just such evidence. As Samuel Butler said, the art of good living consists in the "ability to come to adequate conclusions from inadequate evidence." This applies equally well to the art of good medicine. Immanuel Kant put it more incisively: "Again and again it is necessary to take a decision on the basis of knowledge sufficient for action but insufficient to satisfy the intellect."

One might wish that the evidence for anticoagulant therapy were flawless enough to satisfy the intellect of all clinicians, but it is sufficient, I believe, for therapeutic action.

This discussion of the treatment will be limited largely to the coumarin derivatives. Heparin, although quicker and more potent, does not maintain an adequate anticoagulant effect longer than 16 to 18 hours without repeated injections. Therefore, its usefulness, except for the lipolytic effect, is limited to thromboembolic attacks and to the first few days of acute myocardial infarction. There are many questions that must be answered about antithrombotic therapy:

First: In patients with symptoms of impending myocardial infarction, can early treatment prevent the attack? Presumably this would occur through prevention of thrombosis. The results have been disappointing and certainly not convincing.[7,22,23]

Second: Can anticoagulant therapy prevent propagation of a coronary arterial thrombus that has already occluded a vessel? Theoretically it should, but the available evidence does not support the assumption.[5]

Third: Can it prevent cardiac mural thrombosis? In a number of studies analyzing necropsy data on patients who had had acute myocardial infarction it was concluded that mural thrombosis was prevented in about half the treated group.* The Committee on Anticoagulants noted, though, that "relatively few of the participating physicians gave doses the first three days sufficiently high to achieve optimal therapeutic levels by the fourth or fifth day of dicumarol therapy." This failure in early treatment apparently could account for the failure to prevent mural thrombi in a larger number of cases, and argues for the early administration of heparin as well as dicoumarin. As regards long-term therapy, Bjerkelund's necropsy study (the only adequate controlled necropsy study I could find), disclosed a left ventricular mural thrombus in only one of nineteen treated patients compared with six of twenty-four controls.[4]

Fourth: How is extracardiac thromboembolism affected? The clinical diagnosis of thromboembolism is notoriously inaccurate. The committee observed that venous thrombosis and embolism of the lungs, brain, abdomen and extremities seemed to be significantly reduced, and necropsy data accorded with this conclusion. Among untreated patients, emboli, with or without infarction, averaged 125 per 100 cases, as against 45 per 100 of the treated. The main antithrombotic effects in acute myocardial infarction, therefore, are in the prevention of cardiac mural thrombi and extracardiac thromboembolism.

Fifth: Can continuous dicoumarin therapy prevent recurrent coronary artery thrombosis and reduce mortality? Nichol and Fassett[18] first suggested that it might in 1947. All the long-term studies of survivors of acute myocardial infarction, ranging from two to ten years' observation, have shown a reduction in mortality among the treated group to half or less of the rate in the untreated group. Bjerkelund's study of 119 treated and 118 untreated patients, whom he kept under observation for three to three and a half years, is one of the most carefully controlled of the long-term studies, and is the only one in which necropsy data is available on both groups. He reported a reduction of 45 per cent in recurrent infarction and of 37 per cent in mortality from cardiovascular disease. At necropsy of 43 patients, coronary thrombosis was noted twice as frequently in the control group as in the treated. Suzman, Ruskin and Goldberg,[24] and Manchester[15] found a significant reduction in mortality from recurrent infarction in treated patients observed from four to ten years. Other studies, although not well controlled, have shown a considerably less than the statistically anticipated mortality for two to three years following the initiation of anticoagulant prophylaxis.[12,17,19,25]

Sixth: What are the risks of bleeding? First, it should be emphasized that a risk of serious bleeding is the only justifiable contraindication to anticoagulant therapy. A little bleeding ordinarily cannot kill; a little clotting can. Second, the risk of bleeding incurred by anticoagulant therapy cannot be judged except by comparison with a comparable control group. Third, the details of treatment must be considered. Reports of hemopericardium and other effects of bleeding incurred by overdosage are irrelevant to evaluation of a drug whose contraindications and method of administration are now fairly well delineated. Most reports on serious hemorrhagic

*References 8, 9, 11, 13, 28.

complications reveal such deficiencies of methods.† Intracardiac hemorrhage and rupture are the major hazards from anticoagulant therapy in acute myocardial infarction. In mild infarction this danger is negligible, and in a large series comprising both mild and severe cases it is still small. The committee estimated that in each 100 cases treated, anticoagulant therapy might be expected to cause two more deaths from rupture or hemorrhage than would be expected without this treatment. "This regrettable loss," the committee added, "fortunately is counterbalanced by a substantially lower expectation of death due to thromboembolism." In long-term therapy, the statistical incidence of moderate or severe bleeding has been about once every 13 to 20 years of treatment per patient. Serious episodes have been those of cerebral hemorrhage in patients with hypertension.

DISCUSSION

If one dismisses the radical view (which was expressed recently to me in a personal communication) that since anticoagulant drugs are used to kill rats, they should not be used in man, there are reasons behind the fairly clear-cut areas of agreement and disagreement that exist among clinicians. The available evidence receives varying interpretations as it passes through the thinking man's cerebral filter. Anticoagulant therapy is theoretically indicated in impending myocardial infarction even though its benefit in this situation is not measurable. Probably by the time the thrombotic milieu signals its presence with persistent angina, it is too late in most cases to prevent the impending occlusion, except by rendering the blood absolutely incoagulable. Moderate reduction of coagulability, however, may prevent coronary thrombosis in some cases. Moreover, it should diminish the size of the occluding thrombus and, in any case, if thrombosis occurs, the recommended therapy for the acute attack and for later prophylaxis is already initiated. Most observers who have had long experience with anticoagulants believe that in acute myocardial infarction, under proper conditions, the benefit of the drugs exceed the danger in the "good-risk" cases of low mortality, and even more so in severe cases. Burchell[6] sums up anticoagulant therapy in good-risk cases: "We are willing to give anticoagulant therapy to 100 or more patients with the hope of preventing one death." Long-term therapy, in the general opinion, is most beneficial for older patients with recurrent infarction or thromboembolism, but Bjerkelund[4] found the greatest benefits in patients under 60 years who had had only one infarct. The death rate in such patients was significantly reduced in the first year of observation. He suggested that the energy available for treatment would be most profitably expended on long-term therapy of relatively young patients who have had only one infarct.

TREATMENT

For anticoagulant therapy there is no routine; but there is a method with clear-cut requirements. Success of the method depends, first, on the readiness and competence of the physician to apply it and, second, on the proper selection of the patient. Lack of experience and lack of immediate and reliable laboratory facilities are a definite contraindication which nevertheless can and should be overcome in many cases. Decision as to whether anticoagulants are indicated may be a perplexing one. In some communities and hospitals the majority of physicians, at least the more voluble ones, may so outspokenly oppose anticoagulant therapy in certain circumstances that the attending physician, unless strong in his own conviction, may be constrained to forego use of it. On the other hand (in a situation more frequently stressed) he may be urged toward this therapy by other physicians, by the patient or his relatives or (it is said) by the rather naive fear of being charged with negligence. In either situation he should consult, as he does in other clinical dilemmas, with a colleague also experienced in anticoagulant therapy.

The selection of the patient must not be based on any rough routine criteria, but rather on his specific need and, for long-term treatment, on his willingness and ability to cooperate. At times the indications and the contraindications may be of almost equal weight. Bay and co-workers[3] reported such an instance: A patient who had had several thrombotic coronary occlusions and intercurrently three massive hemorrhages from peptic ulcer, insisted on being given dicoumarin during recurrent thrombosis because he feared the danger of thrombosis more than that of hemorrage. It was decided to administer the drug as the lesser risk. Fortunately the need for such decisions is rare. In an acute case, if no consultant is at hand the attending physician's decision on the use of anticoagulant drugs is unassailable. If the danger of hemorrhage cannot be assessed for lack of laboratory service, or if it seems too great in the circumstances, a frank opinion can be given without fear of reprisal. Long-term anticoagulant therapy is the more demanding. The liaison between physician and patient becomes of paramount importance. Such therapy is not feasible in private practice unless a dedicated secretary and nurse are available, and they must have a bent toward amateur sleuthing in locating wayward patients. It is unwise unless the patient accepts equal responsibility in treatment.

†References 1, 2, 10, 14, 21, 26.

The third consideration—given a competent physician with adequate facilities, given a clinical situation that justifies this therapy—the third consideration is the willingness and ability of the patient to cooperate, to come in for his blood tests, to report any significant experience, to follow the prescribed dosage and precautions and, above all, to keep in touch with the physician's office. Failure in this regard can nullify the benefit of treatment. The patient who at first seems cooperative may later fail in these requirements; and if he does, the physician had best gradually diminish dosage and then completely discontinue the treatment.

These, then, are the requirements for therapy. Are they enough? Will they protect the patient—and the physician—from the disquieting experience of sudden severe hemorrhage? My answer, and that of other physicians experienced with anticoagulant therapy, is yes, within the bounds of reasonable risk. First of all, bleeding is almost never spontaneous. As experience with hemophiliacs, and patients with a total loss of fibrinogen demonstrates, a person with virtually incoagulable blood does not bleed unless he incurs a vascular lesion.[20] Security, then, is in direct proportion to the thoroughness with which the physician seeks and rules out the causes of hemorrhage. Obviously it is impossible to detect, much less to evaluate, every potentially hemorrhagic lesion in the body. As the patient's prothrombin level is reduced, the risk of hemorrhage from such lesions increases. The next consideration, then, is to proportion the risk of hemorrhage to the risk of coronary thrombosis. No arbitrary prothrombin level can always protect against these opposing dangers. In acute myocardial infarction the danger of both cardiac thrombosis and cardiac hemorrhage is higher than in the chronic stage. In the latter, experience indicates that a fairly moderate prothrombin reduction gives good protection against thromboembolism without undue risk of hemorrhage.

The Committee on Anticoagulants believes that to receive the full benefits of anticoagulant therapy, the prothrombin content of the patient's blood should be kept, at all times, at a level indicated by at least 25 seconds for clotting (a value of 23 per cent or less). There is, however, the committee said, a definite but lesser therapeutic effect with a prothrombin time as short as 20 seconds (a value of 40 per cent). "A little anticoagulant protection," according to the committee, "is definitely better than none. In cases where excessive hemorrhage or other medical risk precludes the use of dicoumarin in the usual doses, or maintenance of the usual prothrombin levels, consideration should be given to the use of minimal doses such as would maintain the patient at about 20 seconds prothrombin time." Thus, even with a known cause of potential bleeding, a patient with severe myocardial infarction should not automatically be denied antithrombotic protection. On the other hand, a patient without a bleeding lesion need not be kept at a low prothrombin reduction for long-term treatment. In my experience, a prothrombin level of 40 per cent gives good protection against thrombosis, diminishes the risk of bleeding and permits an interval of several weeks between tests once a stable level has been determined. Until better means are available, then, anticoagulant drug therapy is here to stay. The problems ahead are to sharpen the criteria for patient selection, to improve the techniques in management, and to train more physicians in the method. It is the duty of medical schools now to give students a sound understanding of anticoagulant therapy and to present the areas of agreement and disagreement with an avoidance of bias. Equally, practicing physicians have a duty to become skilled in the technique and to be prepared to use it with confidence.

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Nonverbal Treatment of Neurosis

Techniques for General Practice

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• "Psychosomatic medicine" does not demand that the general practitioner function as a psychiatrist; rather, it is a psychiatric orientation that can increase the effectiveness of purely medical treatment for such conditions as neuroses. The general practitioner to whom the patient turns may achieve permanent results with nonverbal techniques where formal psychotherapy would be impracticable or unacceptable.

The first aim is to relieve pressure so that the patient can regain his mental balance and thereby his self-confidence. Arts, hobbies, sports, and the like can be prescribed rather specifically according to the patient's personality and needs. Nutrition can be improved simply at first by prescribing needed additions to diet rather than imposing restrictions. Vitamin deficiency may by itself be the cause of neurosis or more serious mental disease, whereas psychic stress by itself may create a need for additional vitamin intake. Hormone therapy may be extremely helpful but must be based on clear indication and limited to specific purposes.

Since lack of sleep and rest quickly impairs mental function, it is important for neurotic persons to learn relaxation as a necessity for sleep. Sedatives may be used in a crisis but should be abandoned as soon as possible.

With all drugs there are problems of excess and habituation. The least, the mildest, the shortest dosage is the ideal.

The initial steps of psychotherapy are available to any physician: Establishing rapport, noting how complaints are stated, encouraging ventilation, winning confidence rather than immediate results.

HOWEVER SINCERELY the general practitioner may acknowledge the concept of psychosomatic medicine and the inadequacy of purely physical treatment for disease, he cannot be expected to function as a part-time psychiatrist in addition to his other duties. What is he to do, though, about mild neuroses that are not serious enough to require psychiatric referral or even consultation, but for which sympathetic listening and common-sense advice are not effective? Is he to be content with placebos and topical treatment while mutual discouragement overcomes him and his patient?

This problem cannot be solved by insisting that the general practitioner is the counterpart of the old family physician. The social and economic conditions which were the basis of that close relationship cannot be reconstructed at will and therefore the comparison has at best a sentimental value. But he is the physician who has been consulted, and he can offer help which might not be accepted as readily from a psychiatrist.

Fortunately, a number of techniques have been evolved which may be effectively applied in such borderline cases. These "nonverbal techniques," as they are here termed, may substitute temporarily or permanently for the primarily verbal method of the psychiatrist. Some of them are ancient, some have developed in the same period as modern psychiatry; but all have been overshadowed until recently by the psychoanalytic method and some have almost fallen into disrepute. Now the tide has turned. Nonverbal techniques have assumed a much more important place in the psychiatrist's management of the neuroses, and the general practitioner as well can use them safely.

A twofold change has taken place with regard to what was formerly known as "physical methods" in psychiatry. Chemotherapy and substitutional therapy have been added to the old techniques like insulin and electric shock, lobotomy and leukotomy. These newer methods do not require hospitalization, can easily be combined with any kind of topical treatment, and are particularly indicated in neurotic disturbances; formerly held in lower esteem as mechanistic and merely symptomatic, they are now accorded full status. Meanwhile, better understanding has been gained regarding the role of the patient in working out his own problems and thereby acquiring a working degree of mental health. This process in the patient has assumed increasingly greater importance,[60] and anything which might help it can now be regarded as contributing to the final result. "The actual therapeutic potential is to be found and elicited in the patient, not applied by the psychotherapist."[51] Measures formerly used only to quiet the patient or to keep him occupied (occupational therapy) are now prescribed with a different aim—to help him over a crisis, this furthering his own efforts toward integration: "The patient,"

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Submitted August 2, 1958.

Taylor[48] explains, "is guided either directly or indirectly to realize how much he can do for himself rather than being made to feel how much the therapist is doing for him." Prescribed activity can become "the affirmative manifestation of an otherwise maladjusted nature." Sedation is seen in the same light. The physician's function, says Alexander,[2] is to remove obstacles and thus "create conditions in which the regenerative powers can best act . . . Once an obstacle is removed, the rest of the therapeutic task can be entrusted to the ego. War experiences have shown that in many traumatic neuroses simple sedation may suffice. This allays anxiety that temporarily interfered with the ego's integrative faculty. One is inclined to assume that in procedures like narcosynthesis the major therapeutic effect is the relief of anxiety . . . which temporarily hampered the ego's integrative capacity." Thus sedation is employed with a psychotherapeutic intent. The same applies to all nonverbal techniques in the treatment of the neuroses: Each might be prescribed for merely temporizing or supportive purposes, but by the psychotherapeutic intent they become important means of psychosomatic management available to the general practitioner.

Recreation

Nonverbal techniques of therapy were first applied in the treatment the child, who in "therapeutic play . . . may discharge his feelings without fear of being censured or punished. Such a secure emotional discharge serves to quickly reduce the child's anxiety, enabling him to move safely on to therapeutic experiences."[23] The adult equivalent of children's play—leisure activity—may be used in the management of the neurotic patient, both to amuse him and to promote his readjustment through "the psychodynamics of the highly motivated play situation."[9] The overriding consideration must be to propose constructive use of leisure hours, weekends and vacations, which for poorly organized persons may be emotionally upsetting and initiate regressive behavior.[28]

To this end, the physician himself must be familiar with the wide range of therapeutic and supportive measures. To mention a few: Bibliotherapy has been described as a means of psychotherapy through reading. Patients with some insight into their problems may profit from semi-popular books dealing with mental health,[13,16] but novels, poems or plays likewise may divert the patient and at the same time introduce new ideas and suggest purposeful goals. Music[25] and graphic arts, too, may awaken the patient's creative resources. A group setting is frequently preferable to single instruction or work, and the emphasis should be not on technical proficiency but on releasing suppressed emotions and unexpended energy, and on opening up new fields of interest. It is precisely for this reason that, for a professional artist whose real or neurotic problems arise from his work, art therapy may even be detrimental. For the same reason, less pretentious art forms—Moreno's psychodrama,[59] folk dancing, handicraft and the like—may be more beneficial than the fine arts with their professional orientation. Whatever the hobby, it should be selected on the basis of the patient's life setting, traits and complaint. For instance, it would certainly be wrong to recommend stamp collecting for a person of compulsive tendencies. On the other hand, chess, although a silent and lonely game affording only a minimum of interpersonal relationship, might be for some patients a means of working out deep-seated problems.[27]

Nutrition

More conventional supportive measures, such as are used in many purely medical situations, may be applied to the neurotic patient with the same psychotherapeutic emphasis. The difference not only lies in a changed physician-patient relationship, but extends to follow-up and duration of the prescribed regimen. In treating a medical disorder by diet or by vitamin or hormone supplementation, it is not necessary to prolong the follow-up much beyond the subsidence of acute signs and symptoms; but in the psychotherapeutic situation these improvements must be presented as a new way of life that must be pursued regularly for sustained effect.

The main difficulty with diet is not to determine the best regimen but to select one acceptable to the patient. It may be best to begin with only a few needed additions to his habitual diet. In the minds of many people diet has become a bugaboo, an additional source of frustration, a punitive device denying one of the few reliable remaining means of gratification.[1,33] The strategic approach is to be satisfied initially with slight but effective adjustments in the patient's nutritional pattern. Once confidence is established he will more readily accept further changes until a regimen is worked out on the underlying principle well formulated by Sargant and Slater: Sufficient quantities of a well-balanced diet rather than large amounts of an ill-balanced intake.[41] The transition to a fully adequate diet thus becomes part of an overall learning process.

Vitamin supplementation should be seriously considered for any psychosomatic or neurotic complaint; it is well known that the classical deficiency diseases often are accompanied by mental symptoms.[42] Pellagra, for example, leads in many cases to overt neurasthenia, but many neurasthenic symptoms may occur as the only evidence of vitamin B_1 deficiency, as also in the prepellagrous stages of B_2

deficiency—insomnia, anorexia, palpitation, headache and irritability. The night blindness of vitamin A deficiency often occurs on a hysterical basis. The clinical picture in other vitamin deficiencies is less clearly delineated, but in general when one vitamin is lacking the supply of all others can be presumed to be correspondingly low.

Physicians are at present in the skeptical stage of their experience with vitamins, but better understanding of nutritional factors[21] and more refined methods of experimentation and observation[15] have made it possible to demonstrate convincingly the usefulness of vitamin supplementation in the management of the neuroses. It is recognized, first, that the vitamin content of the ordinary diet is too unreliable to support the stress of psychosomatic disease, and second, that supplementation of one vitamin may even accentuate rather than relieve symptoms. Usually, continuous supplementation is necessary for a long time before vitamin metabolic balance is restored and normal tissue demands consistently met. Like diet, vitamin supplementation should become part of the patient's way of life, to be continued almost indefinitely. In general, any multiple-vitamin preparation, preferably including minerals and other nutrients, may be prescribed in doses sufficient for established minimum requirements. Such therapy has by itself dissipated pronounced neurotic symptoms,[50] and in combination with drug therapy has been found highly effective in a clinical setting comparable to a private office.[12]

Hormone Therapy

Hormone therapy in psychiatry, Sargant and Slater[43] concede, "is rather the hope of the future than the practical measure of today." It should be restricted to recognized indications in demonstrable deficiencies and never abused as a cure-all. Desiccated thyroid, in doses of one or two grains a day, counteracts mild depresion. Confusion, lethargy and inability to concentrate may be the result of myxedema[5] and disappear on administration of thyroid; but it must be remembered that prolonged intake of the hormone can lead to thyroid atrophy. Estrogen, possibly combined with progesterone or testosterone, may be tried in menopausal symptoms as well as in premenstrual tension. In some women, though, estrogens aggravate depression and should not be given over a long period without a thorough endocrinologic evaluation. Most healthy women can sustain the stress of hormone fluctuation, and prolonged or severe symptoms indicate more basic problems.

Rest and Activity

One of the most critical deficiencies is lack of rest and of sleep, in which "the activity of the self-system can be abandoned for a certain part of the twenty-four hours."[47] Lacking this relief, a person's ability to handle unwelcome, disapproved motivations, anxieties, etc., deteriorates rapidly with consequent impairment of mental health.[46] According to Pavlov[32] "sleep is inhibition spreading over all the hemispheres"; this quite generally explains the restorative power of rest, sedation and sleep.[57] These are not the same as the sleeplike conditions of anesthesia and hypnosis[14] and in all probability sleep induced by hypnotic drugs is in this latter category, overpowering somatic tension rather than releasing it.

It is by now well understood that wakefulness is due to anxiety or, more generally, to overactive ego defenses. The aim is to by-pass these defenses in order to alleviate the causative anxiety or other neurotic tendency through the curative effect of healthy sleep. Physiologically, sleep is a state of functional de-afferentation[18]; in particular the feedback from muscular impulses must have come to rest before the patient can follow the diurnal rhythm from wakefulness to sleep. This may be achieved by relaxation of the body in a tepid bath (as the ancient Greeks recognized[26]), by a better planning of the day's activities to permit rest periods, and by a slowing down of pace toward bedtime. One person may be quieted by an evening walk, another by reading, others by prayer[49]; but in any case, "sleep must be wooed and is not to be captured in one swoop."[31] As Kleitman reminds us, sleep is at least partly a learned process.[19] Medication is only used to bridge the first critical period of insomnia or broken sleep; it should be of the mildest effective kind and withdrawn as soon as practicable. Myerson[30] made the excellent suggestion that it should be taken an hour or two before retiring so that by the time the patient gets to bed he is really ready for sleep. Toilet preparations should be completed shortly after the evening meal; thus the patient, instead of relying on drugs, is learning habits conducive to a good night's rest.

Necessary as ample sleep and rest are for the neurotic patient, the management should never become overprotective. Whitehorn[52] cautioned that stress has become a "bad word"' and that we fail to recognize how much mental illness is attributable not to stress but to the lack of it or, in his own words, to "the personal experience of uselessness and a collapse of meaningful effort." A truly productive plan of reeducation should lead to "the organization of life toward energetic effort in stressful situations rather than the avoidance of stress." For some persons, work (for example, gardening, carpentering, mechanics) may be as health-giving as rest is to others. It is interplay between rest and effort that makes for stability through adjustment.

Drug Therapy

The ancient sedatives and stimulants—alcohol, opium, hashish, tobacco, caffeine—have been vastly supplemented in recent years, but the underlying problems of excess and habituation remain the same. We must ask ourselves whether more than temporary relief of neurotic symptoms is ever achieved by these methods, and whether even this result is not too high a price to pay in view of the dangers. Even the tranquilizers have recently been listed as habit-forming drugs by the World Health Organization.[56] The theme of this presentation applies to drugs also: They may serve to help the patient over a crisis from which he can proceed on his own powers. Always, the drug must be (1) selected with regard to the history and symptoms; (2) the mildest agent in the lowest effective dosage; (3) followed up and withdrawn at the first sign of ill effect or when no longer required.

Tranquilizers tend to reduce anxiety, nervous and muscular tension and acuity of awareness; they are mildly depressant, but not as powerful in their action as central depressants like the bromides or barbitals.[22] The best current opinion seems to be that chlorpromazine and reserpine are very useful in the treatment of patients in hospitals, but they are much less promising for ambulatory neurotic subjects. Anyway, their effect is not clearly predictable. For example, chlorpromazine seems to be beneficial in most patients with acute anxiety neuroses, while in others anxiety and related symptoms are decidedly increased.[39] The drug seems to do best in neurosis of the obsessive-compulsive type; in depressive states it is ineffective and may even aggravate the complaint.[24] Reserpine usually relieves neurotic symptoms within a matter of hours, but it is likely to break down protective defenses and release underlying drives in a display of emotions which may be disturbing to the patient and difficult for the physician to handle.[20] Most important, both chlorpromazine and reserpine as well as meprobamate (Equanil,® Miltown®) are beset by a considerable number of untoward side-effects, among which may be mentioned leukopenia, impairment of liver function, urinary retention, purpura, diarrhea and hypotension.[58] In this context the results of a carefully controlled British study are very enlightening.[35] Various tranquilizers ("Nutinal," chlorpromazine, meprobamate, and "Sedaltine") were tested against amobarbital (Amytal) and a placebo; while there was no difference in the potency of the four tranquilizers, only the barbiturate proved significantly more effective than the placebo. Some observers may go as far as to conclude that "in general the tranquilizers appear to have little or no advantage over placebos in the treatment of neurotic outpatients."[10] At any rate, although amobarbital and the other barbiturates are not without hazards of their own, physicians are at least aware of the risks involved and familiar with indications and dosage, whereas even the widely used tranquilizers have not yet been sufficiently tested for anyone to be sure of all their effects.

Stimulants like benzedrine, dextro-amphetamine and others are helpful in the management of slight depressions but cannot control the more severe forms. The effect, furthermore, is short-lived. Sargant and Slater[40] suggested that benzedrine may afford the depressed patient a day or two of comparative normality and that this experience can be used to impress upon him that his illness is not as hopeless as he thought. When anxiety is the predominant symptom, though, stimulants will only aggravate the condition. Myerson[30] observed that a combination of amphetamine derivatives with one of the barbiturates helps to reestablish an approximately normal emotional state, thus bringing the latent forces of the organism for cure or remission into play. Ferguson[12] employed a stimulant (methylphenidate hydrochloride) to counteract the effect of psychosedatives and neurosedatives as well as the patient's initial underactivity, and thereby was able to produce a state of "active tranquility."

The earlier expectations that the ataractic drugs, in addition to exerting a sedative effect, will also increase ego strength do not seem to have been fulfilled as yet.[6] The old barbiturates and bromides, far from being merely "chemical restraints," still remain very useful tools in the management of neurotic crises. Placebos, on the other hand, hardly deserve a place in the treatment of psychosomatic disease. It has been recognized that every drug in addition to its pharmacological effect also exerts a psychological and sociological influence, the latter being due to the "milieu effect."[30] Furthermore, the action of placebos is not entirely imaginary, for measurable physiologic changes at the end organs have been demonstrated.[55] Yet it would seem that in the management of the neuroses the use of placebos introduces an element of deceit which clashes with a basic concept of psychotherapy, that is, the establishment of a relationship of confidence between physician and patient in order to promote healthy reactions in the disturbed subject.

DISCUSSION

The foregoing discussion of nonverbal techniques, although far from complete, may suggest the wide variety of modalities available, their applicability depending on the patient's personality, his resources and the preference of the physician. There is a personal style in nonverbal psychotherapy, as in more formal methods, and the physician can best use the means he understands and relies on. Indeed, all

psychotherapy has important elemental and nonverbal aspects,[8] relating back to the early, nonverbal events with which psychopathology is so much concerned. "The patient has to gain communicative experience in the nonverbal mode before he can engage in verbal exchange,"[37] and communication "motivated by love in the widest sense of the word including the religious, and guided by understanding . . . is capable of producing profound psychic and physiologic effects."[4] This element, moreover, may prove to be the basic ingredient in the management of neuroses, for which the rate of success appears to be similar whatever modalities have been employed. Bowman and Rose,[7] reviewing 11 reports by different investigators over a 20-year period, found the range of improvement from 55 to 87 per cent with an average of 67 per cent. Miles and coworkers[29] arrived at a mean average of 73 per cent, while Schjelderup,[44] in a smaller series, obtained lasting improvement in better than 87 per cent with psychoanalytic treatment alone. Eysenck's tabulation,[11] although widely criticized, suggests at least worthwhile improvement from treatment by custodial institutions and general practitioners (72 per cent) compared with success of psychoanalysis (66 per cent) and eclectic treatment (64 per cent).

As Braceland[8] observed, "We now may regard it as axiomatic that no one approach to psychiatric disorder can claim a monopoly upon wisdom, understanding or therapeutic efficiency." Redlich[36] noted that most psychodynamic therapy is applied to patients of the upper and middle classes, while psychoneurotic persons of lower class are most likely to receive only supportive and manipulative psychotherapy. He ascribed the difference not only to economic reasons but also to difficulties in communication between the middle-class psychiatrist and the lower-class patient. The general practitioner, more accustomed to dealing with patients of all classes, may be better able to bridge the gap both verbally and nonverbally.

Certain psychiatric techniques may be helpful in guiding the physician to a selection of therapeutic methods. The history, first, should be taken in an unhurried manner. The patient who is always under some strain is thus given a chance to relax. This does not mean that the permissive approach is necessarily in all instances the best; sometimes firmness is much more effective, but the physician's basic attitude should be open and truly compassionate, never moralistic or prejudging. What is required is to establish rapport. Only if the patient feels that he can trust the physician will he open up and talk freely, thereby giving the therapist an opportunity to collect the facts and size up the situation. Rapport has been described as a necessary nonspecific factor in any psychotherapeutic procedure,[61] and the same holds true with regard to ventilation: The patient is encouraged to tell his story, thereby unburdening himself of his troubles. Such an emotional release is sometimes all that is required, but in other instances it will at least indicate his readiness to accept constructive measures and suggestions. However, a word of caution may be in order—"not to remove the lid from the boiling cauldron of the patient's emotions unless it can be got back again."[53]

What makes of routine history-taking a preliminary psychotherapeutic interview is the added attention given to every seemingly unimportant detail. It is important not only *what* symptom the patient complains about, but *how* he does it. Simple questions as to age, family status, occupation, general health and former diseases often bring forth answers which reveal the degree of adjustment to essential life situations. From all these data together with the patient's general appearance and manner the physician may form an impression of his personality and the seriousness of his condition—most important, his accessibility and his insight into his problems. On these findings the physician will decide how best to manage the case or whether a psychiatric consultation might be indicated.

Not only at the time of the first consultation but throughout the treatment by nonverbal means, the physician can find occasion for verbal contact with the patient. In fact, one of the greatest advantages of these nonverbal methods is that it becomes possible to weave psychotherapy unobtrusively into other consciously acceptable treatment methods, thereby enlarging the range of communication. Many a patient who cannot be reached by the verbal approach will be helped by such nonspecific therapy.

As in every other kind of psychotherapy, the immediate aim is relief of symptoms, and only time can tell what further benefits will accrue to the patient. Results will differ, of course. In some cases the subject will again be able to function on his former level, whatever that level might have been; in others there will slowly emerge a greater ability to cope with life's problems, a change for the better which the subject enjoys without fully understanding what has happened; or again, improved surface adjustment might lead to greater insight, more conscious re-learning, and resolution of deep-seated conflicts. Psychiatry has come to be much more realistic in its therapeutic goals and not to expect a complete remaking of every patient.[17] Thus, whatever the response may be, the general physician can make an important contribution by helping the patient to use his own recuperative powers for a fresh start toward better mental health.

The list of references is available from the author.

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Crushing Injury of the Hand

Prevention of Ischemic Contracture

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• **Crushing injury of the hand usually causes "explosive" damage. Subsequent swelling of the palmar structures further impairs venous outflow, and hemorrhage into structural spaces increases the pressure. The arterial system and the large dorsal veins, however, are seldom obstructed and provide adequate circulation unless hampered by improper bandaging. A bandage that compresses the dorsal veins causes back-pressure, which increases the swelling further and brings about ischemia. Swelling and pain cause the patient to restrict exercise of the injured hand, which permits contractures to develop.**

The author has averted this sequence in more than 100 cases by preserving integrity of veins during debridement, arresting hemorrhage, bandaging the hand with compression dressings in functional flexion, and reducing swelling with hyaluronidase. In these cases, on removal of bandages in 24 hours, swelling was reduced and continued to diminish. All patients exercised the hand at this time without discomfort and only a few required aspirin for pain.

VOLUMES HAVE been devoted to reconstruction of the hand crippled by contractures after injury, but little has been written about methods of immediate treatment to prevent or lessen impairment of function during the healing process. This presentation describes a method which in the author's experience has greatly reduced the severe effects of intrinsic ischemic contracture, a major cause of deformity and disability in crushing injuries of the hand.

Bunnell[1] believed that ischemic contracture is due to vascular impairment, often caused by tight encasement of the hand in plaster or in constricting bandages. He distinguished this condition from Volkman's ischemic contracture of the arm but recognized that the two might coexist. Both, he postulated, are due to the same cause—not arterial obstruction, which causes gangrene at the fingertips, but local congestion or impairment of venous return.[2] He distinguished this ischemic contracture of the intrinsic muscles from the typical claw-hand resulting from median and ulnar nerve paralysis. He stressed the importance of recognizing vasomotor disturbances early and of keeping the hand active and working; early flexion of the proximal finger joints and dorsiflexion of the wrist, he mentioned, maintain muscle balance of the hand in functional position.[3]

VASCULAR SUPPLY OF THE HAND

On the back of the hand a rich, large network of veins lies directly over the unyielding extensor tendons and fascias covering the metacarpal bones, but the loose skin allows free flow under most conditions. Dense networks drain the fingers, then unite as a dorsal plexus and converge at the elbow as two large veins, the cephalic and the basilic. Smaller trunks proceed proximally between these two.

The palm of the hand allows no such freedom of circulation. It is a basin, bounded by the bony metacarpals, the intrinsic muscles and the thenar and hypothenar structures. The superficial palmar veins, small in caliber, sparse and irregularly distributed, lie between the skin and the palmar aponeurosis. The deep veins follow the digital arteries, the common digital arteries and the superficial and deep palmar arches to the ulnar and radial arteries. The common digital vessels and the nerves form neurovascular bundles coursing between the flexor tendons and lying beneath the deep palmar fascia. As on the back of the hand, all the vessels converge at the wrist.

Presented before the section on Orthopedics at the 87th Annual Session of the California Medical Association, Los Angeles, April 27 to 30, 1958.

PATHOLOGY OF VENOUS OBSTRUCTION

Despite the freedom of the dorsal veins of the hand to expand, trauma and the resulting unnatural pressures can obstruct them within seconds.

Bunnell said: "Venous obstruction promptly brings about engorgement with blood. Soon capillary resistance is overcome at 60 mm. of mercury, and at 100 mm. petechiae and extravasation of blood commences, accompanied by edema. In a closed space even arterial inflow is reduced, regulated only by the amount of outflow."

Brooks found that venous occlusion causes hemorrhage, edema and degeneration of muscle fibers, resulting in acute inflammation progressing to fibrous contracture. He described more inflammation than degeneration but did not test it in a closed space. He concluded that venous obstruction is the primary cause of the condition.

In the operating room the author has observed that within a very short time after crushing trauma the hand begins to swell; it may double in thickness in 10 to 20 minutes. Crushing causes an "exploding" kind of injury characterized by ragged and irregular skin lacerations from which extrude muscle bellies, torn veins, fascia and areolar tissue. There may be little or no bleeding. Soft tissues appear edematous. On deep exploration, rents in the palmar fascia are seen, sometimes welling free blood from hematomas of the palmar clefts. Flexor tendons may appear intact but on closer inspection contusion may be seen. Areolar tissues may be torn loose from the neurovascular bundles. Peripheral nerves, because of their resistant composition, almost always escape division, but if they are compressed against firm structures such as bones, the resulting hyperemia and edema may cause partial or total anesthesia for a period of weeks to months.

Crushing injuries affect the palmar circulation of the hand more seriously than the dorsal circulation. Seldom are the arteries severely damaged; they usually pulsate fully and regularly, although moderate transient arterial spasm sometimes occurs. On the back of the hand, where contusion is often severe even though accompanied by little or no laceration, the veins are usually thick-walled and movable enough to roll away from the impact and avoid serious injury. The dorsal skin being elastic enough to permit heavy swelling, the severance of even a small dorsal vein leads to extensive subcutaneous hematoma, but even despite edema the dorsal veins usually remain patent. In the palm, however, superficial veins are torn because of their vulnerable position, and the deeper veins because they are thinner and more friable. Injured veins early become obstructed or thrombosed. As the structures bounding the palmar basin swell with edema, the pressure increases to a point at which the deep venous flow is partially or fully occluded but arterial function is not restricted.

In this situation the dorsal superficial veins become vitally important as the only remaining channel for venous return. The slightest dorsal pressure exerted by constricting bandages or casts at the wrist dangerously slows or completely halts the already overtaxed flow, establishing the conditions for venous ischemia—unobstructed arterial pressure, severe venous insufficiency, and blockage of available venous outlets. In such circumstances the hand becomes thick, boggy, purplish, moist and cyanotic, in contrast to the dry gangrene of arterial occlusion. The results—fibrosis and contracture—are known to all.

The multitude of permanently crippled hands suggests that basic principles of treatment, if understood, are not being thoughtfully applied. Potential ischemia of the crushed hand must be averted in primary treatment; procrastination in the belief that secondary reconstruction will suffice should be condemned. Prophylactic, specific medical and surgical therapy immediately following the injury is imperative whenever possible. In the author's experience, these are the conditions that assure good salvage:

Proper debridement; control of hemorrhage; maintenance of adequate venous and lymphatic flow; arrest and reduction of edema; relatively painless motion.

In debridement, functioning veins must be respected. Too often, in the removal of foreign material and devitalized tissue, veins are cut and tied to clear the operative field with little regard for their importance. Rather they must be meticulously teased free and gently retracted while the operation is carried deeper. They can best be dissected without tourniquet occlusion, since if they are filled with coursing blood they are easier to identify and isolate than if they are collapsed. Rarely are ties indicated. Severed, bleeding vessels need be clamped only a few minutes until adequate thrombosis has taken place, when the forceps can safely be removed. After identification and retraction of the venous plexus the tourniquet can be reapplied.

All bleeding points must be sought, identified and clamped, the hemostat including only the end of the cut vessel. Even the smallest of bleeding vessels can flood a hollow space and increase interpalmar pressure. Large masses of tissue indiscriminately clamped and tied become necrotic and add to edema and to accumulations of waste products in an already overtaxed area. Ties should be kept to a minimum and should be made with 6-0 plain catgut or finer. Actually only the larger arteries need be ligated. Residual hemorrhage anywhere in the hand, especially in closed spaces, can be disastrous. No reliance is to be placed on the use of pressure dressings or closing sutures to control active bleeding.

Adequate venous and lymphatic flow is maintained by preservation of the venous plexus as previously described. Proper "compression" bandages are vital for good circulation. Much has been said and written about this subject but improper bandaging is still one of the major causes of local venous ischemia. Bandages placed with maximum pressure across the wrist obstruct the venous and lymphatic outflow. Casts and improperly secured plaster splints have the same effect. Placing the arm in a sling compromises the circulation and in many patients adds deltoid atrophy.

After operation, open wounds should be closed by interrupted stainless steel No. 38 wire sutures. The edematous tissues should be approximated loosely, for tight closure increases intrapalmar pressure and

retards circulation. Wounds are covered by Furacin® or Vaseline® gauze. Compression dressings are formed by the use of fluffed Kerlix® gauze, two to three yards as indicated, and placed to fill the palm like a large ball so that the fingers and wrist assume the position of function about it. Another fluff is placed on the dorsum of the hand. These fluffs must not overlap the wrist. Wrapping with stockinette bandage cut on the bias is started over the metacarpophalangeal joints fairly snugly so that the fluffs are compressed about one-half. The pressure is decreased as wrapping progresses to the wrist so that at this point an examiner's two fingers can be slipped loosely between the last turn of bandage and the volar or dorsal wrist skin. A volar splint then can be placed and fixed to the arm and hand by a loose stockinette bandage cut on the bias, but it must be ascertained that this does not alter the pressure of the underlying bandage. The arm is not slung. The patient is instructed to carry his hand at upper chest level and to exercise his shoulder, elbow and fingers immediately.

The measures already discussed help to arrest and to reduce edema. Fairly recently a new agent that can be used for this purpose, hyaluronidase, was introduced. Literature concerning hyaluronidase is meager, especially on its use in trauma. Gartland and MacAusland suggested that the enzyme may disperse hematoma from traumatic soft tissue hemorrhage and thus prevent impending Volkman's contracture or possible myositis ossificans if 1,500 turbidity reducing units are injected into multiple sites and pressure applied with an elastic bandage.[4] It is generally conceded that the enzyme promotes diffusion and consequent absorption of fluids in the tissues. No evidence was found that it might cause localized infection to spread, provided it was not injected into the infected area. The author has infiltrated hyaluronidase directly into the debrided soft tissues about the site of reduced and pinned fractures, nerve repairs, muscle and fascial suture, with no complications or delay in healing. The amounts used were 150 units in fingers, 200 to 300 in thumbs and 600 to 800 in multiple areas of debrided viable tissue of the hand before the application of the pressure bandage.

The foregoing routine has been followed successfully in more than 100 cases. The author regularly removes bandages within 24 hours and inspects the hands. In all cases there has been a definite diminution of edema and swelling, the reduction continuing during the ensuing 5 to 14 days, until the hand appears essentially normal.

Early motion of the hand promotes maximum recovery; restriction of motion tends to cause crippling. If moving the hand causes much pain, the patient of course moves it as little as possible; and pain cannot be adequately controlled, in the author's experience, by narcotics or barbiturates. Rather, the measures previously outlined have proven to be effective insurance against pain of motion. Most patients were made comfortable with aspirin. Some did not require it. The first postoperative day none needed narcotics, few needed aspirin to make them comfortable, and 95 per cent reported only mild discomfort. All exercised the fingers and thumb freely without undue discomfort. Most returned to work two or three days after operation, on jobs requiring only the uninjured hand.

CASE REPORTS

CASE 1. A 19-year-old paper jogger caught his left hand between steel and rubber rollers, which crushed the fingers and hand before he could be extricated. The wound was highly contaminated with bits of paper and other foreign material and almost immediately swelled grossly (Figure 1). It was of the explosive type caused by compression and extended from the dorsum of the thenar eminence across the palm to the hypothenar eminence (Figure 2). Almost all the superficial palmar veins were severed but there was little gross hemorrhage. Soft tissues, portions of muscle bellies of the hypothenars, fat and connective tissue extruded from the wound. The deep palmar fascia was likewise irregularly and intermittently exploded, and the flexor tendons of all four fingers were exposed and contused, though intact. Their accompanying neurovascular bundles were intact but the surrounding areolar tissues were torn and partially stripped away. Some of the common digital veins appeared thrombosed and insufficient, but the arteries pulsated normally and fully. Petechial hemorrhages were apparent everywhere, with small hemorrhages in the hollow spaces. Roentgenograms showed no fractures.

After debridement, hemostasis and closure of dead spaces, the wound was closed loosely with stainless-steel wire sutures (Figure 3) and 800 units of hyaluronidase was infiltrated directly through the wound. The fingers were flexed about a fluff of Kerlix and a volar plaster splint was applied over Kerlix and bandage to hold fingers and thumb in a position of flexion (Figure 4). This was retained with a layer of stockinette (Figure 5). Antitetanus serum and Combiotic®* (2 cc.) were injected.

The operation had been done under local nerve block, and the patient was then sent home with a supply of aspirin for moderate pain and Empirin Compound 3® to be taken in case of severe pain. Twenty-four hours later edema had regressed and the palm of the hand was again concave. The patient had taken only 1.2 gm. of aspirin for pain and had been able to sleep. He reported only a slight degree of pain on examination. The postoperative course was uneventful and since a one-hand job was avail-

*A combination of crystalline procaine penicillin G, buffered crystalline potassium penicillin G and dihydrostreptomycin.

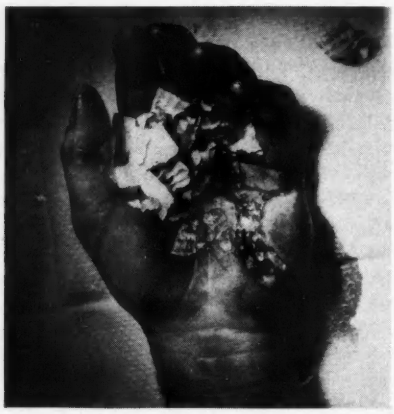

Figure 1.—Swelling and contamination in hand crushed between rollers of paper-jogging machine (Case 1).

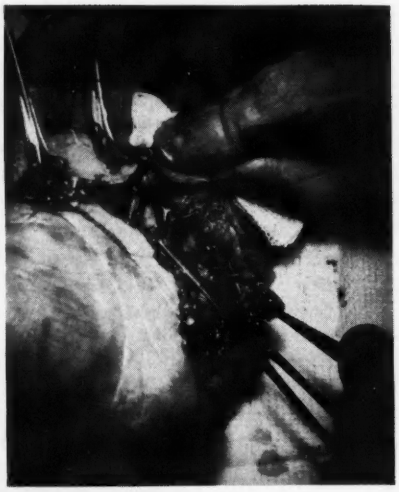

Figure 2. — Same hand as at left, showing wound of explosive type extending from dorsum of thenar eminence across the palm to the hypothenar eminence.

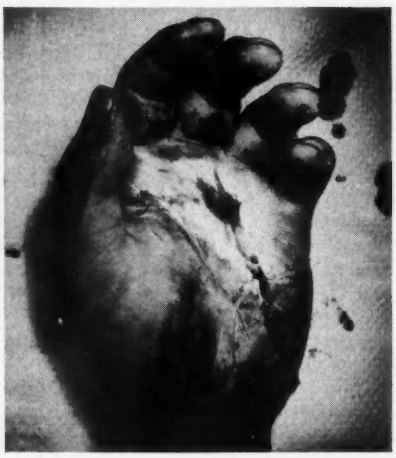

Figure 3.—Same as above, after debridement, hemostasis, closure of dead spaces and loose closure with stainless steel wire.

Figure 4 (Case 1). —Hand flexed over a fluff of Kerlix and bandaged to hold fingers and thumb in flexion.

Figure 5. — Stockinette holding hand in position shown in Figure 4. Note that it is not tight at wrist, lest circulation be impaired.

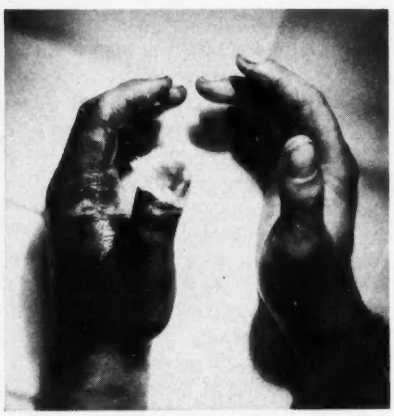

Figure 6 (Case 1).—Injured hand 24 hours after operation, with edema diminished and palm concave.

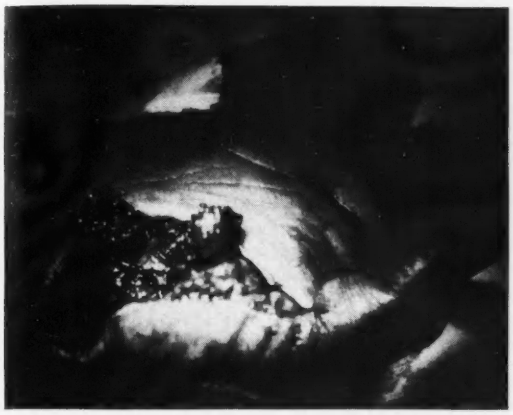

Figure 7 (Case 2).—Bursting laceration principally at base of thumb caused when hand was crushed in a press.

Figure 8 (Case 2).—Ten days after injury, fingers and thumb could be almost fully extended.

Figure 9 (Case 2).—Flexion of fingers and thumb of injured hand ten days after injury. Adduction and abduction of thumb were somewhat limited.

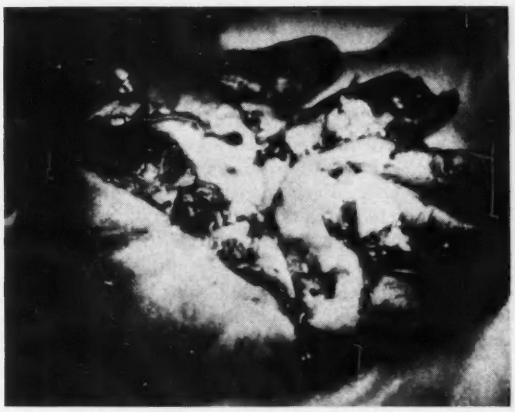

Figure 10 (Case 3).—Hand crushed and almost amputated when caught under falling bundle of steel sheets.

Figure 11 (Case 3).—The thumb and little finger, in which digital arteries and dorsal veins still functioned were salvaged.

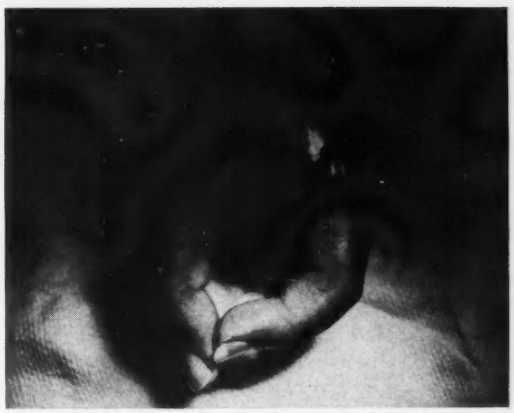

Figure 12 (Case 3).—A useful pinching function was developed by the patient six months after operation.

able he returned to work. Three weeks later he resumed his regular job. Permanent disability was minimal.

CASE 2. A 35-year-old man caught his right hand in a press, suffering a bursting laceration principally at the base of the thumb (Figure 7). Treatment previously outlined was followed. A large hematoma was evacuated through a stab wound. Twenty-four hours later the edema had regressed so that the dorsal skin was loose and wrinkled with the hand in full, painless extension. Ten days later the fingers could be almost fully extended and flexed (Figures 8 and 9). The thumb could be flexed and extended well, but adduction and abduction were more limited. Meantime the patient had returned to a one-hand job with loss of only a day from work. Permanent disability was minimal and in six weeks the patient went back to his regular job.

CASE 3. The left hand of a 32-year-old Negro was crushed and almost amputated when a half-ton of steel sheets fell on it (Figure 10). Multiple open comminuted fractures included the extrusion of two carpals through the wrist. Tissues of the index, middle and ring fingers as well as the central palmar area were obviously devitalized and were excised, but a spark of life remained in the thumb and little finger, in which the digital arteries and dorsal veins still functioned. Fractures of these digits were reduced and stabilized with intramedullary Kirschner wires, so that they could be formed into a pinching mechanism. Hyaluronidase was infiltrated into the little finger (150 units), the thumb (300 units) and the proximal palm and wrist (800 units). Closure, medication and compression bandaging were done as previously described.

The fractures and soft-tissue injuries healed per primum, and so did a full-thickness skin graft applied six weeks after operation. In six months the patient had developed a useful pinching function (Figures 11 and 12).

CASE 4. An 85-year-old woman's hand was caught in the wringer of a washing machine, which avulsed the skin and subcutaneous tissues like a glove from the wrist to the finger bases. Both the dorsal venous plexus and the deep palmar vessels remained substantially intact. There was little damage to the deep palmar fascia and the extensor tendons. Treated as previously described, the patient was discharged from hospital the day after injury with little edema and very little pain, moving her fingers freely. Three days after operation the skin was healing and the patient was using the hand in sweeping, etc. Six months later she could almost fully extend and flex the fingers.

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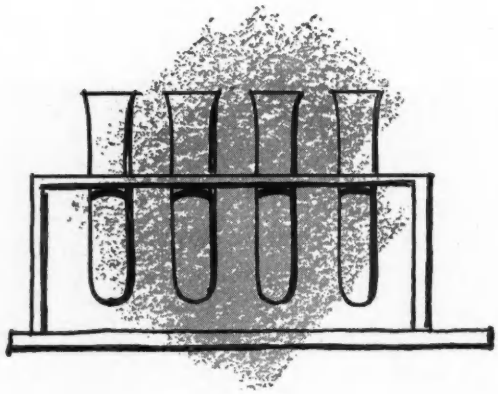

Congenital Heart Disease

Comments Regarding Incidence and Natural History

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• **The incidence of congenital heart disease is approximately 1 per cent of all live births. Approximately 60 per cent of patients who die of congenital heart disease do so at less than two years of age. Very few patients with such lesions live beyond 45 years at the very most. In about 70 per cent of patients who are born with cardiac anomalies, the lesions are either of kinds that are already being operated upon successfully or for which operations are now being attempted and often are helpful.**

IMPROVEMENTS IN THE diagnosis and treatment of patients with congenital heart disease have come swiftly. Even investigators working full time in the field find it difficult to keep up with the frequent advances. Oddly, however, little is known about prevention and until very recently not much was known about the incidence or the natural history of the various forms of congenital heart disease. Even now this information is fragmentary and hardly permits more than a few general statements.

Estimates of the frequency of congenital heart disease which are generally quoted have been based either upon clinical signs elicited during physical examination of older children, or upon autopsy data, a source which makes hospital deaths from all causes the only basis for comparisons. Richards and co-workers,[10] at Babies Hospital, Columbia University, studied the frequency of congenital heart disease in an unselected newborn population produced from 5,964 consecutive pregnancies. Table 1 shows the incidence of congenital heart disease for the 6,053 infants, grouped according to weight at birth. Fifty of them were born with congenital heart disease, an incidence of 0.83 per cent. This proportion may be slightly low since the living infants were observed for only one year; and it was pointed out that in some cases heart disease probably was "overlooked because of failure to develop symptoms or signs before the end of the follow-up." Richards said that it is reasonable to suppose that atrial septal defects may comprise the major portion of the lesions unrecognized under one year of age.

With this information as a base regarding the incidence, what is the natural history of patients with congenital heart disease? When do they become ill, and when do they die? The classic work in this regard is that of Abbott.[1] For no practical reason at the time, she set about collecting data on cases in which autopsy was done. In an excellent monograph she summarized the material she had gathered on the first 1,000 cases. Survival data on the most common congenital cardiac anomalies (as abstracted from her monograph) are given in Table 2.

It is known from at least two sources[6,9] that 60 per cent to 80 per cent of patients dying with congenital heart disease do so during the first two years of life. Recently, Boesen[4] of Denmark collected data on 1,145 infants and children under four years of age who died of congenital heart disease in all of Scandinavia. The original material has been rearranged by me into three groups: Those in which the lesions were operable, those in which operation was a possibility, and those in which operation cannot be done at present. As shown in Table 3, about a third of the patients had lesions of a kind for which corrective or palliative operation is of proved value.

TABLE 1.—*Incidence of Congenital Heart Disease (CHD) in 6,053 Newborns (Richards[10])*

Weight of Newborn	Total	With CHD	
		No.	Per Cent
500 gm. or less	314	1	0.3
500 to 2,500 gm.	597	18	3.0
2,500 or more	5,142	31	0.6
Total	6,053	50	0.8
Surviving 1 month	5,530	38	0.6

TABLE 2.—*Survival of Patients with Congenital Heart Disease (Abbott[1])*

Condition	No. of Cases	Age		
		Maximum	Minimum	Mean
Atrial defect:				
Above	10	64 yr.	6 mo.	34 yr.
Below	18	46 yr.	8 mo.	19 yr.
Patent ductus arteriosus	92	66 yr.	2 wk.	24 yr.
Ventricular defect	50	49 yr.	Fetus	14 yr.
Coarctation of aorta:				
Adult	70	92 yr.	3 yr.	33 yr.
Infantile	9	9 mo.	8 hr.	2 mo.
Pulmonary stenosis	16	57 yr.	4 yr.	18 yr.
Tetralogy of Fallot	85	60 yr.	11 days	12 yr.

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Submitted July 25, 1958.

TABLE 3.—*Cause of Death Under 4 Years of Age (1,145 Cases);* Operable Lesions*

Diagnosis	Cases	Age at Death Less Than			
		30 Days	150 Days	1 Year	4 Years
Coarctation of aorta	83	40	70	80	3
Patent ductus arteriosus	70	24	54	65	5
Tetralogy of Fallot	48	15	24	36	12
Atrial defect (secundum)	35	10	26	31	4
Atrial defect (primum)	13	1	7	11	2
Aortic stenosis, valvular	30	14	25	29	1
Tricuspid stenosis	30	11	21	28	2
Atrial defect + patent ductus arteriosus	28	19	26	27	1
Pulmonary stenosis	19	9	11	16	3
Total	356	143	264	323	33

*Adapted from Boesen and Vendel: VIII International Pediatric Congress.

TABLE 4.—*Cause of Death Under 4 Years of Age (1,145 Cases);* Operation Possible*

Diagnosis	Cases	Age at Death Less Than			
		30 Days	150 Days	1 Year	4 Years
Ventricular defect	148	51	107	142	6
Ventricular defect + patent ductus arteriosus	65	34	54	63	2
Ventricular defect + atrial septal defect	15	1	10	14	1
Ventricular defect + patent ductus arteriosus + atrial septal defect	12	5	10	12	
Pulmonary stenosis + atrial septal defect/ventricular septal defect/patent ductus arteriosus	33	13	24	29	4
Atrioventricular communis	33	6	22	33	
Complete transposition	167	78	138	163	4
Single ventricle	90	52	80	88	2
Total	563	240	445	544	19

*Adapted from Boesen and Vendel: VIII International Pediatric Congress.

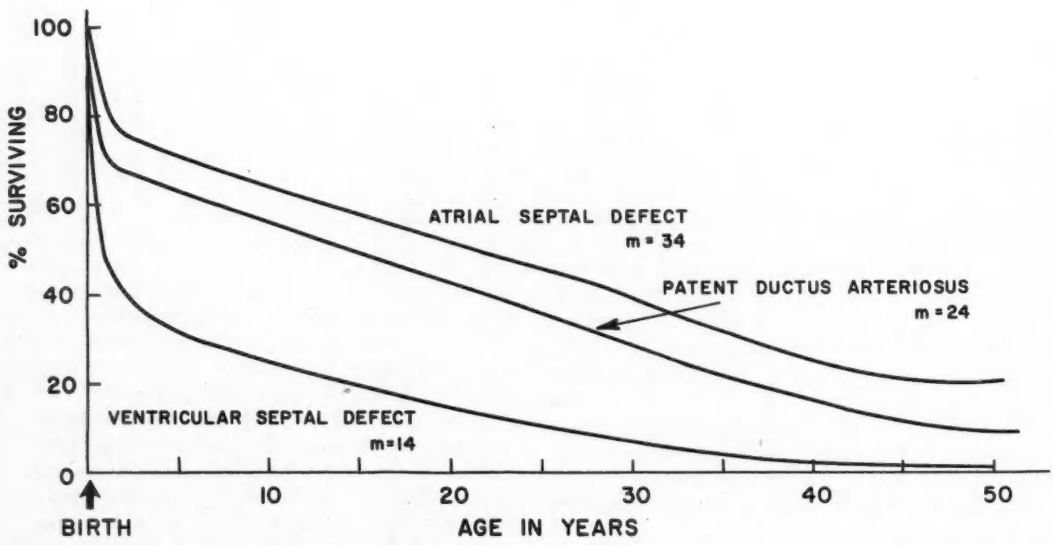

Chart 1.—Theoretical survival curves for patients with left to right shunt (mean age (m) obtained from Abbott[1]).

About half of the 1,145 patients were in the possibly operable group—that is, they had lesions for which operation is now being attempted and is either curative or palliative in many instances.

In order to visualize the natural history of some of these congenital heart lesions, I have made two diagrams which are purely theoretical but make use of the best information currently available. Chart 1 shows the theoretical survival curves for patients with left to right shunts and Chart 2 shows the curves for patients with stenotic lesions.

Congenital heart disease is not only significant in the cause of death in infancy but it is also a significant cause of morbidity. Keith[7] recently reviewed his experience in the diagnosis and treatment of heart failure in the pediatric age group. He reported on 304 patients, of whom 90 per cent were under one year of age. Chart 3 shows the causes of heart

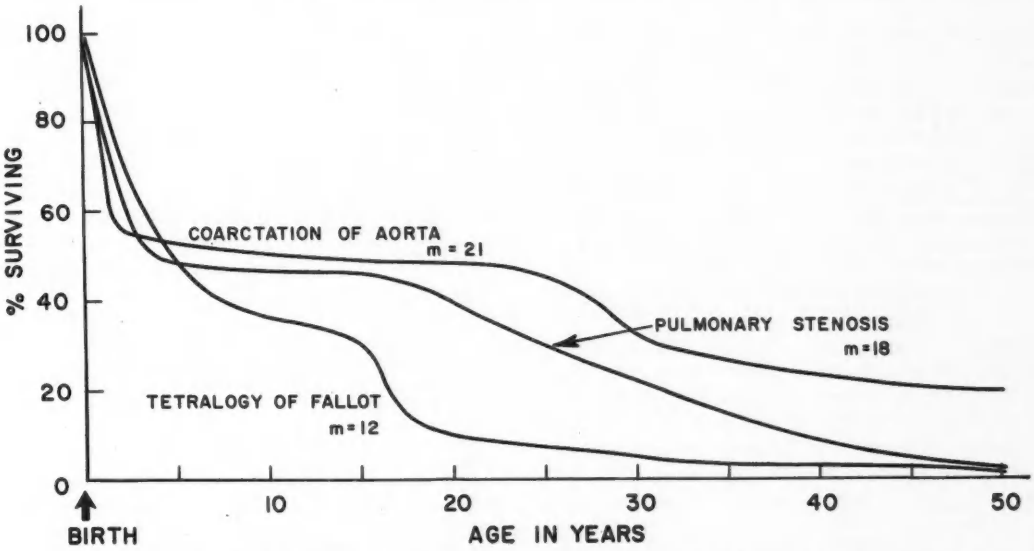

Chart 2.—Theoretical survival curve for patients with stenosis (mean age (m) obtained from Abbott[1]).

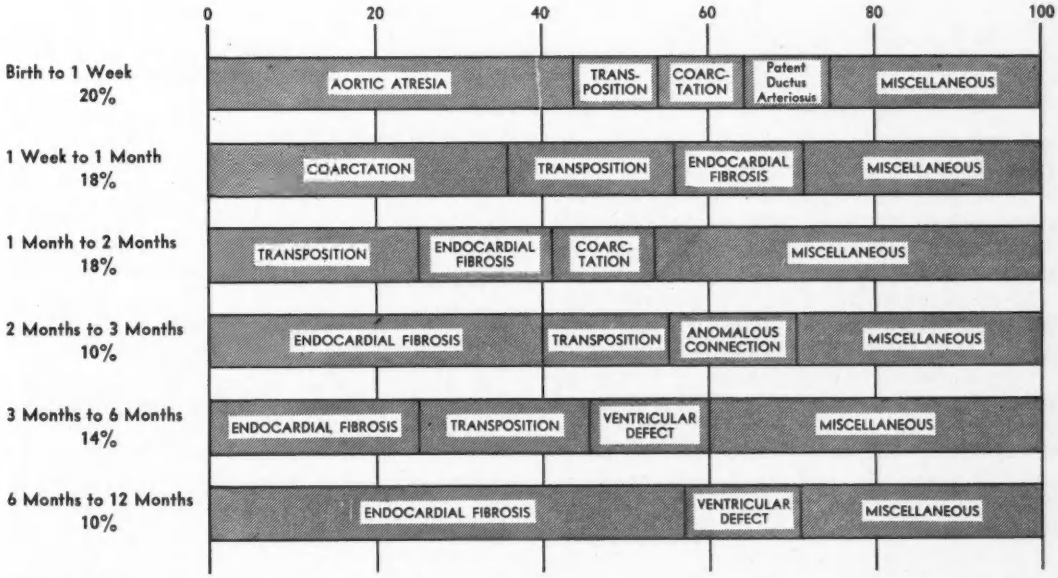

Chart 3.—Cause of heart failure correlated with age (304 cases). (Adapted from Keith: Pediatrics, 18:491, 1956.)

TABLE 5.—*Cause of Death Under 4 Years of Age (1,145 Cases):* No Operation Available at Present*

Diagnosis	Cases	Age at Death Less Than			
		30 Days	150 Days	1 Year	4 Years
Hypoplasia of left ventricle	63	43	60	63	
Truncus communis	48	33	42	47	1
Hypoplasia of aorta	42	21	36	39	3
Diverse	73	22	47	68	5
Total	226	119	185	217	9

*Adapted from Boesen and Vendel: VIII International Pediatric Congress.

failure in patients under one year of age. In addition to the high incidence of transposition of the great vessels and coarctation of the aorta as a cause of heart failure, the high incidence of endocardial fibroelastosis, particularly after the first month of life, was noteworthy.

It is becoming increasingly evident that in the instance of at least two congenital heart lesions, patent ductus arteriosus and interventricular septal defect, there is a far-advanced clinical situation in which surgical treatment by present methods is probably contraindicated. In the severe form of "reverse" patent ductus arteriosus, in which the major portion of the flow through the ductus arteriosus is from pulmonary artery to aorta, the operative mortality is approximately 100 per cent, for reasons not well understood.[8] In contrast, in cases in which the major portion or all of the flow through the ductus arteriosus is from aorta to pulmonary artery, the operative mortality is below 1 per cent.[11] Recently evidence was presented to suggest that patients with a partial "reverse" ductus arteriosus operated upon at an early age can survive the operation even though there is some reverse flow.[3] The main problem is early recognition and definitive treatment.

Eisenmenger complex has long been recognized as a form of interventricular septal defect. The main aberration that distinguishes it from simple interventricular septal defect is a systolic pressure in the right ventricle which exceeds that in the left ventricle, causing right-to-left interventricular shunt.[5] Most of the patients with nearly balanced pressures in the right and left ventricles have extensive changes in the intima and media of the pulmonary arterial tree.[5] Evidence recently presented[2] showed that the operative mortality in this group of patients also approximates 100 per cent, whereas it is much lower for patients who do not have the elevated right ventricular pressures.[2] Clear-cut evidence is not available to show that right ventricular and pulmonary hypertensions progress with time. However, severe forms of the disease with pulmonary hypertension are known to exist in the infancy and early childhood period. It is entirely possible that the pathological changes present in the lungs of younger patients with the disease will not be so extensive or irreversible at this earlier age.

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Criteria for Mitral Valvotomy

Roentgen Evidence in Pulmonary Hypertension

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• **Although catheterization is the most accurate and sometimes the only adequate method of measuring pulmonary hypertension as an indication for mitral valvotomy in rheumatic heart disease, it is so costly and complex that simpler methods are desirable. Clinical evidence of pulmonary hypertension is least accurate; electrocardiography is confirmatory in half of all cases. Roentgenologic findings are more helpful; moderate or severe enlargement in the pulmonary arteries has been associated in 92 per cent of cases with resting systolic pressure of 50 to 90 mm. of mercury in the pulmonary artery. In cases in which there is little or no enlargement, hypertension may still be present and demonstrable only by catheterization. Other roentgen signs noted as helpful are abrupt narrowing of the large branches of the pulmonary artery in the middle and lower lobes, and the septal lines of Kerley.**

MAXIMUM IMPROVEMENT following mitral valvotomy has been obtained among those patients in whom pulmonary arterial pressure was highest before operation and fell the most afterward. Cournand and Ferrer[4] separated patients with classical mitral diastolic murmur into three groups. In the first group, those with mitral stenosis but normal pulmonary vascular pressure and insignificant degrees of mitral "block," valvotomy is useless. In the second group, where mitral stenosis is complicated by active carditis, with reduction of cardiac output and only a slight increase in pulmonary vascular pressure, operation is contraindicated. Only in the third group, those with true mitral block and varying degrees of pulmonary hypertension, is the operation desirable.

Although cardiac catheterization may adequately distinguish among these groups, its cost and complexity are deterrent; but since pulmonary hypertension is so important a criterion for mitral valvotomy, it is important to assess it by other means—clinical, electrocardiographic or roentgenologic.

Purely auscultatory criteria of pulmonary hypertension are not sufficiently reliable; as Fowler[6] and others have stated, the signs of this condition cannot be clinically measured with accuracy. Fowler specified that the following signs are of little value for the purpose: (1) The loudness of the apical diastolic murmur, (2) the prominence of the second sound in the pulmonary area, (3) the presence or absence of a right ventricular "lift" along the left sternal border, and (4) the presence of an "A" wave in the venous pulse.

The senior author[2] observed better correlation with pulmonary hypertension in the electrocardiogram, which yields a characteristic pattern of right ventricular hypertrophy for half the patients with increased right ventricular and pulmonary artery pressure. In eight of eleven patients in Fowler's series who had mean pressure of more than 50 mm. of mercury, there was electrocardiographic evidence of right ventricular hypertrophy, and in the other three there was suggestion of this condition. Among those with pressure between 30 and 49 mm., hypertrophy was evident in five, suggested in one; in four there was no such evidence of abnormality. Ordinarily, commissurotomy would have been considered indicated for all these patients. Rarely was right ventricular hypertrophy electrocardiographically evident in a patient with mean pressure below 30 mm.

The purpose of this presentation is to describe the roentgenographic findings in pulmonary hypertension. Jacobson and co-workers[8] observed that moderate or severe enlargement in the pulmonary arteries was associated in 92 per cent of the cases with moderate to severe increase of resting systolic pressure in the artery—50 to 90 mm. of mercury and higher. However, normal size or borderline enlargement in the pulmonary arterial system does not rule out mild or even moderate pulmonary arterial hypertension, which exceeded 40 mm. in 47 per cent of such cases (see Figure 1). These results agree with those of Steiner[10] and of Doyle and Goodwin.[3] Cardiac catheterization for the sole purpose of determining pulmonary arterial pressure is probably unnecessary when enlargement in the pulmonary artery is moderate or marked. When there is little or no enlargement, however, pulmonary hypertension can be excluded only by cardiac catheterization.

Doyle and Goodwin[3] also emphasized the abrupt narrowing of the large branches of the pulmonary

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Presented before the Section on Internal Medicine at the 88th Annual Session of the California Medical Association, San Francisco, February 22 to 25, 1959.

arteries, a condition they observed both at necropsy and in postmortem angiograms. This abrupt narrowing was present, they noted, in the arteries to the middle and lower lobes, as would be expected with a general increase in peripheral resistance, but there was no such narrowing in the arteries of the upper lobes. They advanced the opinion that the arterial constriction, both reflex and organic, represents an

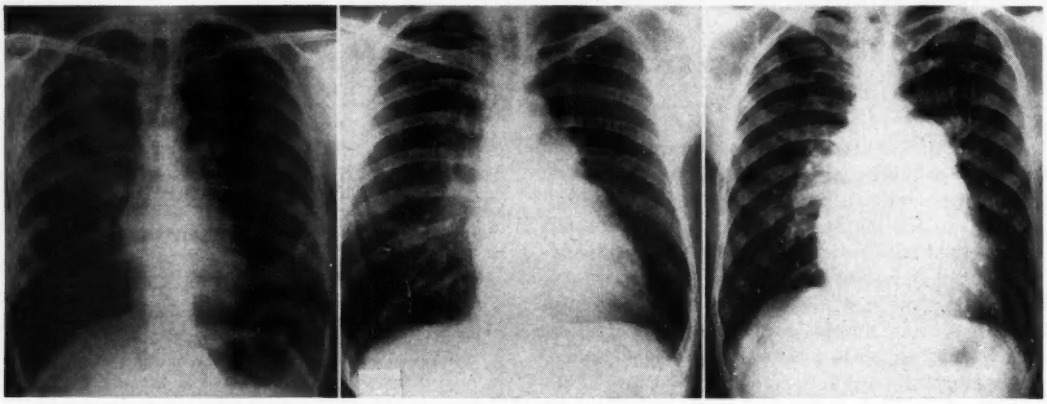

Figure 1.—The size of the pulmonary artery and its relation to pulmonary hypertension. *Left:* Normal pulmonary artery with normal major branches in a case of mitral stenosis with mild pulmonary hypertension (40 mm. of mercury). *Center:* Moderately enlarged pulmonary artery with slightly enlarged major branches in a case of mitral stenosis with moderate pulmonary hypertension (78 mm.) and mild hemosiderosis. *Right:* Greatly enlarged pulmonary artery and moderate enlargement of major branches in a case of mitral stenosis with severe pulmonary hypertension (200 mm.). The multiple calcifications in both lungs are presumably related to previous rheumatic activity but their presence cannot be correlated with pulmonary hypertension.

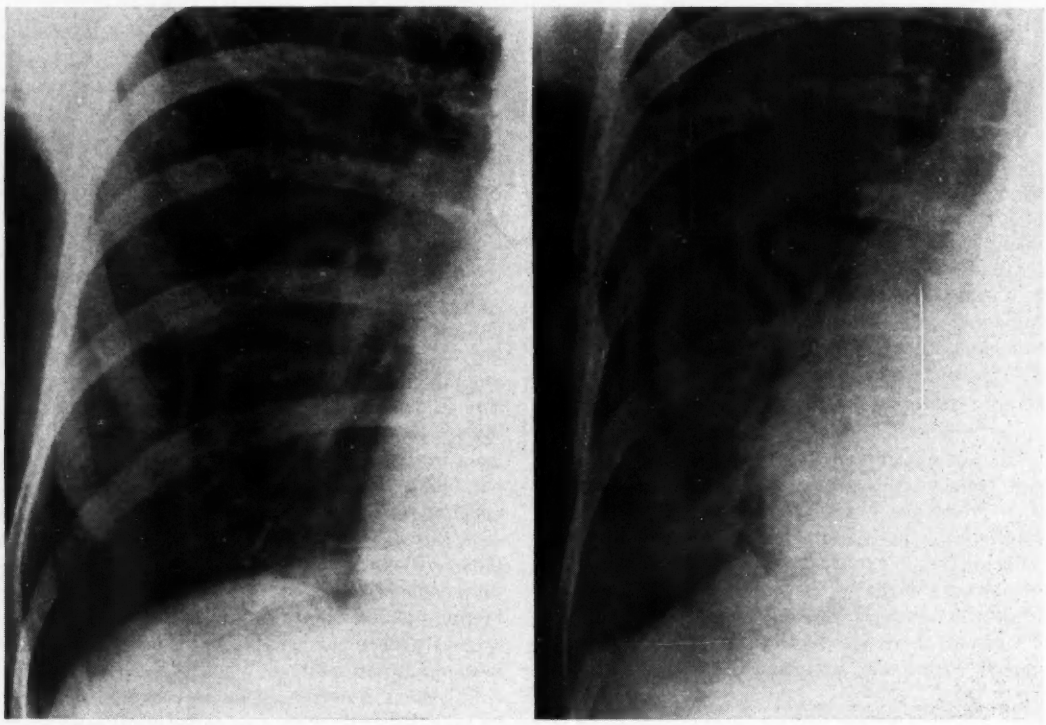

Figure 2.—X-ray films in a case of long-standing mitral stenosis. No septal lines present in the film at left. In the picture at right, three years later, after development of mild pulmonary hypertension and recurrent congestive failure, numerous septal lines are visible.

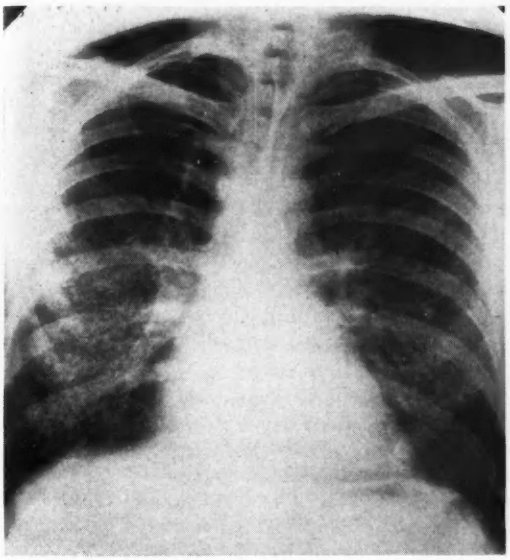

Figure 3.—X-ray evidence of extensive hemosiderosis with normal pulmonary artery pressure (proved at operation).

attempt to "protect" the capillary circulation, and that it does not occur in the upper lobes because pulmonary capillary pressure is lower there. They associated this distribution of arterial pressure with the distribution of pulmonary edema in mitral stenosis, which is rare in the upper lobes and greatest in the lower.

The septal or B lines first described by Kerley[9] in 1955 are frequently associated with pulmonary hypertension. These are fine, hairlike, horizontal lines found in both costophrenic angles, more often on the right (Figure 2). They most likely represent distended lymphatics within the interlobular septa and, according to Fleischner and Reiner,[5] may be augmented by hemosiderin deposits. They are found most frequently in patients with mitral block simply because the conditions under which they occur are most often present in mitral valve disease. They are occasionally present in left-sided congestive heart failure, but not in primary cor pulmonale.

Attempts have been made by various investigators to relate the presence of septal lines to increased pulmonary artery pressure. Grainger and Hearn,[7] for example, found that septal lines were present in 14 of 18 patients with diastolic pulmonary artery pressure over 40 mm. of mercury. They further noted that after commissurotomy 90 per cent of the patients with septal lines had improvement, but only 61 per cent of patients without septal lines. Bruwer and co-workers[1] noted septal lines in 30 per cent of 152 patients with mitral stenosis, and in 50 per cent of those who had high pulmonary artery pressure. Although the investigators cited above have attempted physiologic correlations in terms of pulmonary artery pressure, the basic relationship is to pulmonary venous pressure.

Pulmonary hemosiderosis, like septal lines, is most often associated with rheumatic mitral stenosis, and is apparently the result of old hemorrhage at sites of anastomosis between bronchial and pulmonary circulation. Much less common than septal lines, and present in only 8 to 10 per cent of all patients with mitral stenosis, it does not appear to have any specific correlation with mitral block, and is found with all degrees of pulmonary artery pressure (Figures 1 and 3).

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Workmen's Compensation Act

Some Aspects of Interest to California Physicians

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This article was prepared by Mr. William M. Whelan, Director of Special Services, California Medical Association, under the supervision of Dr. Francis J. Cox, Chairman of the Medical Services Commission of the Association, and Mr. Howard Hassard, the Association's Legal Counsel. It is intended as a brief synopsis of the California Workmen's Compensation Law as it applies to the physician in private practice. It is not an exhaustive treatment of the subject. A physician who desires to acquaint himself in detail with California industrial practice should consult the article entitled "The Physician's Role in Workmen's Compensation," CALIFORNIA MEDICINE, 82:352-362, April, 1955. Inquiries regarding industrial medicine should be addressed to Mr. William M. Whelan, California Medical Association, 450 Sutter St., San Francisco 8.

THE California Workmen's Compensation Act was designed to place upon industry the responsibility for treatment and compensation arising from occupational injuries and diseases. Under it, an employer may be a self-insurer or may purchase necessary coverage from an insurance carrier.

The two broad areas of payment made are: (a) Those for medical care to restore the injured employee to his job as quickly as possible, and (b) those cash payments made to the injured employee to replace, in part at least, wages lost while convalescing, or for permanent disability.

During the five policy years 1952-1956, the following incurred losses have been reported in California:

Policy Year	Medical	Indemnity
1952	$ 31,908,676	$ 52,536,772
1953	32,139,841	49,805,739
1954	34,415,465	50,774,849
1955	38,397,186	56,224,907
1956	42,623,506	62,441,190
	$179,484.674	$271,783,457

The Workmen's Compensation Act is administered by the Industrial Accident Commission. The commission is composed of seven commissioners, one of them designated as chairman, who are appointed by the Governor with the approval of the Senate for terms of four years. The commission is divided into two panels of three members each, representing (1) the general public, (2) labor, and (3) management. One panel is located in San Francisco and the other in Los Angeles. The commission employs sixty-seven "referees" who hear disputed cases referred to the commission. During the past fiscal year, the commission rendered decisions involving 33,858 cases. The majority of these cases did not involve the compensability of the claim but, rather, the determination of the amount of the award.

The commission has adopted an official minimum medical fee schedule, which has been published and is available for each member of the California Medical Association. As a matter of convenience, a copy of the printed medical forms used for reporting treatment is to be found at the back of the printed fee schedule.

An employee with an industrial injury is entitled to receive all medical, surgical and hospital treatment essential to the cure or relief of the effects of the injury. This includes medicines, nursing care, transportation expense incident to treatment and examinations, orthopedic aids and other items. An industrial injury is one which, (1) occurs in the course of, and (2) arises out of a given employment. When injured, the employee must notify his employer.

By law, the employer and his insurance carrier have full responsibility for providing required medical treatment and have the right to select the physician and hospital to be used. Although the Industrial Accident Commission administers the Workmen's Compensation Act, the actual control of medical treatment is left to the employer and the insurance carrier. As a matter of practice, the

carriers maintain a register of physicians who have agreed to handle industrial cases.

In an emergency, an employee may get treatment from his own physician. Most employers and insurance carriers will pay that physician for his services at the published minimum rate. Before a physician can look to the insurance carrier for payment, he should assure himself that the injury is an industrial one and should have the name of the employer. Usually, the employee will present to the physician a written notice naming the company for whom he works and the insurance carrier. If the employee does not bring a written authorization stating the name of the carrier, the physician should notify the employer directly. Otherwise, the employer may have no knowledge of the injury and may not submit an employer's report. Carriers will not pay medical bills or compensation until they have received both the employer's and the physician's reports.

The physician who attends an injured employee must file, within five days after the injury, a complete report with the Division of Labor Statistics and Research, Post Office Box 965, San Francisco 1, California. The report must be made on Standard Form 5021. As a practical matter, this form is usually prepared in duplicate and a copy is sent to the insurance carrier. For uniformity, physicians making reports are required to use the standard terminology set forth in the booklet *Evaluation of Industrial Disability* prepared by the California Medical Association and the Industrial Accident Commission.

If continuing treatment is necessary, Supplemental Reports are required. These are a necessary part of the process by which an employee is paid weekly compensation in appropriate cases. In complicated and prolonged cases, Progress Reports are needed, and in certain cases Consultant Reports of examination are submitted. When a case is closed, a Final Report is submitted.

If a physician feels that the fee provided in the schedule is not adequate in a given instance, he should submit supporting data to the insurance carrier to explain his charge. He should note such matters as necessary time spent and should describe procedures and treatment in language that will be understood by nonmedical persons in an insurance claims office.

The commission does have power to arbitrate disputes between a physician and an insurance carrier relative to the amount of the physician's fee. It can assume jurisdiction only after a physician files an application for adjustment of claim. Filing such a claim must be done in a somewhat formal manner and it leads to a hearing. The commission's decisions in such cases are based on general principles of reasonableness. As a practical matter, this procedure would be used in unusual cases.

When an employee files a claim with the IAC, written medical reports, if they are necessary, will be filed by the employee or his attorney as part of the claim. X-ray films should be filed with the commission only upon specific orders (subpoena) to do so. (One of the commission rules provides that if a willful suppression of a medical report is shown to exist, *it shall be presumed* that the findings contained therein would be *adverse* if produced.) Copies of such reports must be served on all parties to the dispute. All medical reports made *during the pendency* of such a claim must also be filed with the commission and a copy served on all parties. When x-ray films are ordered to be produced they must be made available for inspection at a convenient time and place to the party making the request. Hospital records are subject to the same rules. The filing of these reports is the responsibility of the employee or the employer, not the physician.

In most instances, a physician will not be called to testify; his written report is used. But if he *is* called, provision ordinarily will be made for his testimony to be given at a convenient time. In case a postponement is necessary, fair consideration will be given to the emergency appointments of a physician in arranging for a specific time for continuance.

California Labor Code Section 131, provides that a physician subpoenaed to testify before this commission is entitled to the same witness fee and mileage as he is entitled to when he is subpoenaed to appear before the civil courts as a witness. Most insurance companies and employers recognize that a physician should be compensated and they will make provision for doing so.

The commission may refer disputed medical questions arising in compensation proceedings to its own medical bureau or to an independent medical examiner chosen by the commission from an official list of physicians. The cost of the independent medical examination shall be borne by the party requesting it unless the commission orders otherwise.

The physicians listed on the IAC register of independent medical examiners are chosen from among the physicians in the various communities in sufficient number to meet the commission's needs. They are generally specialists.

A medical examiner appointed by the commission *when called for oral examination beyond his written report* shall be paid a witness fee by the party requiring attendance. The fee shall be a minimum of $50 for the first two hours or less of attendance at the hearing, and $18 additional for each hour or fraction thereof beyond the first two.

The Periodic Health Appraisal of Employees

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• **Periodic health appraisals for employees in business and industry are desirable and effective. Male executives over the age of 40 constitute the group most in need of them. Examinations of the kind done for private patients, including careful history-taking and thorough physical observation, are the most reliable, economical and productive.**

Undesirable factors to be guarded against in these examinations are unnecessary hospitalization, excessive laboratory and radiologic procedures and absence of rapport between examiner and examinee.

Ideally, the results of the examination should be made known to the employee only—not to the employer except with the stated permission of the employee.

INDUSTRY AND MANAGEMENT have become increasingly aware of the frequency of premature death and disability in the 40 to 60-year-age group, especially among their male executives.* A number of factors have contributed to the current interest: Articles in the lay press have emphasized the importance of preventive care, and President Eisenhower's illnesses and periodic health appraisals have done much to focus attention on the importance of preventive medicine.[11] Apparently, stressful industrial situations and the increased tempo of life contribute to the hazards of this age group[4]—the years in which executives are most valuable to their companies. For this reason, several large corporations are pioneering in the field of periodic health examinations for executives and other key personnel.[7,9,10] The program of periodic health appraisals is gaining such impetus that unless members of the medical profession (1) crystallize their thinking in this field, (2) formulate sound principles for the health appraisal programs, and (3) make concrete recommendations concerning these examinations, the entire program may fall into complete disrepute.

Commonly, these examinations are of four types:[6] (1) Evaluation of a person in a hospital; (2) clinic evaluation; (3) examination of the kind given by the home town internist, and (4) evaluation by the medical director of the company.[8] In the past, examination by the company medical director has often been relatively superficial, for one reason because full cooperation of the person examined is frequently hard to secure owing to the implication that results of the examination are to be passed on to the company. Not only are hospital and resort evaluations unnecessarily expensive from the standpoints of time and money, but detailed information obtained in these examinations is not immediately available to the employee's physician when necessary.

Years of experience in this field have taught the authors that the most important element in the establishment and continuance of an effective periodic health examination program is the maintenance of such a program on a voluntary and confidential basis.

We shall discuss here only the kind of examination we believe to be the most rewarding—periodic examination by the home town internist. The great majority of physicians would agree that the medical problems of the seriously ill patient can best be handled by beginning with that kind of examination. It follows, therefore, that the most effective way to approach the problems of the potentially ill but ostensibly well person is through the same kind of examination.

We believe that the periodic health appraisal should be approached in the precise and methodical manner used in the private practice of internal medicine. As in the examinations of other patients, the core of the periodic appraisal is the careful, complete history and a thorough physical examination. On the basis of the history and findings in the physical examination, we can determine which laboratory procedures are essential for thorough appraisal of the particular patient. Certain tests are necessary in every instance if we are to assure ourselves and the person examined that important physiologic processes are within normal limits. These are: (1) A complete blood cell count, with hematocrit; (2) determination of erythrocyte sedimentation rate; (3) complete urinalysis, concentrated to 1.018 if possible; (4) serology; (5) stool examination for parasites and occult blood; (6) x-ray examination of the chest, and (7) an electrocardiogram.[5] Additional laboratory procedures are employed only if specifically indicated by a careful evaluation of the patient's history and findings in the physical examination.

One serious and valid criticism of the periodic health appraisal programs of today is the frequent excessive dependence upon exhaustive laboratory

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*References 1, 2, 7, 8, 9, 10.

and radiologic procedures which make the examination costly and tedious but add little to the efficacy of it.[3] Indeed, they may even detract from the value of the health appraisal, because they divert attention from and tend to obscure central problems. A return must be made to careful history-taking and physical examination followed by indicated laboratory procedures.

A fact to be borne in mind at all times is that these examinations should be entirely voluntary on the part of the employee, and that if it entails anything that is not acceptable to him, employee participation in the program will thereby be decreased. One area of controversy is the routine inclusion of sigmoidoscopic examinations in periodic health appraisals. Although medical literature is replete with reports of discovery of unsuspected lesions in routine sigmoidoscopy, the public is not yet conditioned to accept this measure as part of the periodic examination. Rather than insist on the inclusion of this procedure or any other which might materially disturb participation in the program, we feel that it is wiser to perform special examinations only when indications are clear-cut—just as we do in private practice with our other patients.

If possible, health appraisals of this kind should be carried out in the employee's own community. There is much to be gained in permitting the family physician to perform the examination, provided he is qualified to carry out the kind described, since his knowledge of the patient's health history, daily habits and family problems provides a background or setting otherwise unavailable for the evaluation of current findings. This permits a thorough examination which is economical as to both time and money. The physician-patient relationship already built up between the employee and his physician reassures the employee that the results of the appraisal will remain entirely confidential. If the family physician is not equipped to perform these examinations meticulously, they can be accomplished most effectively by a local physician trained in internal medicine. Using local examiners permits better continuity of medical care should the employee become seriously ill, for the information in the health appraisal is immediately available. Providing the employee with an opportunity to use a physician of his own choice emphasizes the voluntary and confidential nature of the examination and promotes the feeling that the examination is a benefit granted by the employer.

All examinations should be conducted in an orderly manner—with fixed appointments promptly met, for this will impress the physician as well as the employee with the importance of the appraisals. Evaluation of approximately a thousand cases of this type has convinced us that the employee should be dealt with exactly as is a private patient.

Once the examination and any indicated special procedures are completed, we insist that the patient return for a review of our observations and for our recommendations. The return visit following the appraisal has a two-fold purpose: It gives us an opportunity to arrange for further procedures as indicated and to set the date of the next appraisal; and the patient is able to discuss the results of his examination and to air any existing anxieties.

Ideally, no report of any kind should be supplied to the employer. We prefer to evaluate our findings orally with each patient at the time of the return visit. Written reports should be given to the employee or to the family physician only if the employee specifically requests that this be done. If the physician, the employee and the employer are agreed that a report should be supplied the company, this report should be initialed by the employee or, better, given to him for transmittal to the employer.

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California's Alcoholic Rehabilitation Program

A Preliminary Report

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• **Confronted by the most serious alcoholism problem in the United States, California has begun an extensive rehabilitation program in three categories: Treatment and rehabilitation services; study and investigation; education, information and training. Treatment clinics operate in seven cities and it is proposed to help finance further facilities in communities interested in their local alcoholic problem. Grants to medical schools for improved personnel training are also proposed. Basic and clinical research and epidemiologic, sociologic and follow-up studies are in progress. Public education to the problems of alcoholism, stressing that it is an illness requiring treatment, is under way. This multi-faceted approach is expected to supply answers to many of the questions surrounding alcoholism.**

ALCOHOLISM is a chronic behavioral disorder manifested by repeated drinking of alcoholic beverages in excess of the dietary and social uses of the community and to an extent that interferes with the drinker's health or his social or economic functioning.[3] Jellinek has devised a formula whereby the number of "alcoholics with complications"—those who have a diagnosable physical or psychological change due to prolonged excessive drinking—can be estimated from the reported deaths due to cirrhosis of the liver. Using this formula, the number of alcoholics with complications in a given community can be determined; and this number, multiplied by 4, gives the total number of alcoholics, the aggregate of those with and those without complications. Field studies of sizable populations to determine the actual number of alcoholics in several geographic areas coincided to a remarkable degree with the number estimated by using the Jellinek formula.[6] No such studies have been carried out in California.

In 1955, according to the Jellinek Estimation Formula, California had nearly 600,000 alcoholics with and without complications or one in every 14 adults in the state, the highest rate in the nation.[1] This was an increase of 77 per cent in the number of alcoholics in the ten-year period since 1945, a period during which the population of the state was rising only 47 per cent. A study made in 1950 showed that the 13 American cities with the highest rate of alcoholism included six in California, with San Francisco and Sacramento ranking first and second nationally.[4] Alcoholism is the eleventh cause of death in the state. ("Alcoholic deaths" include those deaths reported as due to alcoholism and those due to cirrhosis of the liver attributable to alcoholism as computed by the Jellinek formula). In 1955, 87 per cent of all alcoholic deaths were due to hepatic cirrhosis. For years cirrhosis of the liver has been the fifth cause of death in San Francisco.[2]

Approximately half of all arrests in California are for drunkenness. In 1955, drivers who had been drinking were involved in 17,546 accidents and were responsible for 22 per cent of all automobile accident fatalities. In that year the per capita consumption of absolute alcohol for the population, aged 15 years and over, in California was 2.36 gallons compared with 1.90 gallons nationally. In San Francisco the per capita consumption was 4.57 gallons.

In 1954 the California Legislature established an Alcoholic Rehabilitation Commission to study the problem and to institute corrective measures. In 1957 the commission was abolished and its program transferred to the State Department of Public Health, which organized a Division of Alcoholic Rehabilitation. The division administers the program upon the recommendations of a five-man advisory committee representing the public interest. The author is the medical member of this committee.

The program as it now stands—present activities and plans for the future—may be divided into three broad categories. These are: Treatment and rehabilitation services; study and investigation; education, information and training.

Treatment and Rehabilitation Services

The legislature authorized eight "pilot clinics" which were "to engage in the treatment and rehabilitation of alcoholics." There are now seven such clinics in California providing out-patient treatment for alcoholics—in Sacramento, Stockton, Oakland, San Francisco, San Jose, Los Angeles and San Diego—and a new one, in Long Beach, is proposed. With the exception of the San Francisco clinic, which is financed in large part by the county, they are almost

Presented at the Southern Divisional meeting of the American Psychiatric Association, Miami Beach, Florida, December 1 to 3, 1958.

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Submitted December 7, 1958.

entirely state-supported. Some clinics are attached to hospitals, others are in downtown office buildings or in converted houses in residential areas.

Each clinic is headed by a physician, usually a psychiatrist, and has a staff of physicians, psychologists, social workers and nurses. While the Division of Alcoholic Rehabilitation defrays the expenses of the clinic, the local governing body, either the Department of Public Health or Department of Institutions, has a relatively free hand in its operation. The form of treatment rests with the clinic director and may include individual or group therapy, physical rehabilitation, use of drugs, individual or family counseling and vocational assistance. Each clinic keeps detailed records of everyone treated, and the patients will be observed periodically afterward to ascertain the degree of adjustment brought about.

To get adequate data on their effectiveness, it is planned to continue these pilot community clinics for a five-year period, the state paying the cost of operation. In addition to these pilot clinics, it is proposed to offer to supply money on a 50-50 basis to any community that wishes to begin a treatment program—whether in clinic, hospital, "half-way house" or jail farm—or that wishes to provide information centers, counseling services to industry or consultation services to agencies.

Since alcoholics probably seek medical help at some time during their illness, it is proposed to improve the teaching about all aspects of alcoholism in the medical schools in California. Each school will be given a grant of money to establish a treatment clinic for alcoholics. The emphasis will be on the training of medical students, interns, residents and other professional personnel to better their understanding and ability to handle these sick people.

During the past year the Division of Alcoholic Rehabilitation has had two hospital demonstrations that acutely ill alcoholic patients could be treated readily in a general hospital. A corollary study indicated that if the medical, nursing and administrative personnel are given preliminary knowledge about alcoholism, their prejudices and fears abate, which helps the program of treatment go smoothly.

Study and Investigation

The program as it is being carried out embraces basic research, clinical research, sociological studies, follow-up of treated patients and an epidemiologic study. In basic research, studies were made on alcohol metabolism in the body. It was found that alcohol interferes with nutrition in the brain, that it impairs the normal functioning of the pituitary gland and the lower brain stem. The use of tranquilizing drugs combined with alcohol causes greater intoxication than does alcohol alone. The effect of alcoholism on adrenocortical function is under study. Improved methods for the measurement of alcohol in the body have been devised.

In a follow-up study of 700 patients some 12 to 18 months after they were treated a state hospital, a jail farm or a community clinic, it was noted that approximately two-fifths of them appeared to have made a satisfactory adjustment as measured by employment, family relationships, drinking patterns and state of health. The remainder continued to manifest illness by numerous hospital admissions, arrests and patterns of living that brought them into conflict with society. These patients will be observed and interviewed occasionally over a five-year period. The ultimate purpose of these studies is to develop and apply evaluation methods to determine what constitutes good and poor adjustment in alcoholics who have been treated.

Unlike evaluation studies, which are focused on known alcoholics, etiologic studies are focused on the "normal" population to observe the early emergence of problem drinking which may lead to chronic alcoholism. A large number of community leaders were asked to list their criteria of what constitutes an alcoholic. Using these social definitions and criteria, a screening questionnaire was developed which, when applied to any group, permits classification of each member as one of high or of low risk in terms of problem drinking. It is planned to use this screening questionnaire on sizable population groups and to interview persons in both classifications at annual intervals for five years. The hypothesis is that patterns similar to those of known alcoholics will develop in persons in the high risk group. Members of the low risk group will be studied to learn what factors in their backgrounds, personalities and mode of life enable them to cope with alcohol without trouble.

The relationship of hepatic cirrhosis to alcoholism has long been a question. In California, selected death certificates listing cirrhosis are being studied for any evidence of relationship to alcohol. The medical history and pathological slides are being reviewed where possible. Recently it has been found that anatomic and physiologic damage to the liver may be measured in terms of zinc lost from the body fluids. Zinc is a trace metal whose presence is absolutely essential for the functioning of the enzymes which metabolize alcohol. The division is now engaged in a pilot study to validate these findings and then to ascertain their applicability as a simple laboratory test to determine directly the presence of even minor liver damage associated with drinking.

For the past two years a statistical study has been made of death certificates to learn what illnesses alcoholics have at the time of death. To determine possible statistical relationship of various factors, data is also being gathered on the race, sex, occupa-

tion, age and geographical location of the person who died. Mortality tables that are being developed show that deaths due to alcoholism are occurring at younger ages. In 1955 alcoholism was the sixth cause of death for the age group 35 to 54 years. Another aspect of the same study is a scanning of all death certificates for the names of alcoholics who are under observation in this program. When a death certificate is registered for one of these persons, his record is reviewed. From data thus obtained, it will be possible to compute how much shorter the life expectancy is in this group of alcoholics than in the general population.

At an alcoholic research clinic, financed in large part by the Division of Alcoholic Rehabilitation, operating in the department of psychiatry at the U.C.L.A. School of Medicine, the results of treating alcoholics with drugs (including lysergic acid), psychotherapy and placebos are being investigated. Psychological testing indicates that an alcoholic who seeks help at a clinic is similar in personality makeup to the average patient who seeks psychiatric help in an out-patient department, although with more awareness of his rebellion against authority. Other studies, both psychiatric and physiologic, are providing considerable data on the makeup of alcoholics, and on the possibilities of treating them by various means.

Education, Information and Training

The division is engaged in disseminating information on alcohol, on drinking and on alcoholism with a view to encouraging public acceptance of the concept that alcoholism is an illness and that alcoholics are sick people who can be helped. Through workshops, institutes and postgraduate training, professional workers can be educated in the nature, cause and treatment of alcoholism. Physicians, hospital personnel, clergy, police, teachers and social workers would be the groups at whom this training is directed.

COMMENT

From earliest recorded history mankind has been troubled by the problem of alcoholism. The Bible has 71 references to the use of alcoholic beverages. About 2225 B.C. the Babylonian Code of Hammurabi devoted several sections to regulations on the sale of alcoholic beverages to control intemperate use.[5] In 3,000 years of recorded Chinese history, laws for the control of drinking were enacted—and repealed—at least 41 times. Through the centuries, alcoholism has been considered a moral weakness; only lately has it come to be regarded as an illness.

As yet no satisfactory method for the cure of alcoholism has been found. However, no disease in man that affected so many people has ever been controlled by treatment alone. Whether the ancient plague or more recent diseases like malaria, tuberculosis, poliomyelitis or vitamin deficiencies are considered, the ultimate aim is prevention. Now we are beginning to think of prevention in heart disease, cancer and alcoholism. For this we need an epidemiologic approach to determine the causative factors by identifying the life pattern related to the disease—that is, to ascertain the common characteristics of persons who have it and compare them with characteristics in persons who do not.

The epidemiologic approach involves a knowledge of the entire spectrum of alcoholism and a screening device to identify alcoholics in the early stages of the disease. Then it will be necessary to learn what factors enhance and what factors retard the development of the condition. In the case of alcoholism one thinks of such factors as family and marital relationships, employment ambitions, work experience, religious and cultural aspects. While a great deal is known about excessive drinking in the general population, we have no idea of the drinking habits of normal persons. For this reason there are high hopes that the screening device which identifies persons with a high potential for alcoholism and those with only a slight risk will provide a wealth of invaluable data. With it we can learn the pattern of alcohol usage in the general population and can contrast the drinking habits of various categories of persons.

It is hoped that the division's experience in California in the next five years will provide considerable information about alcoholism, much of it new. Some of the information looked for will deal with favorable and unfavorable factors in the treatment of individual patients; the possibility of biochemical, hormonal or other physiological aspects involved in the disease; the relationship of alcoholism to cirrhosis, mortality, crime, divorce and other antisocial behavior; improved testing techniques, psychological, sociological and physiological; and a clearer idea of drinking habits in the general population.

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CASE REPORTS

Hodgkin's Disease

A Report of Two Cases of More Than Twenty Years' Duration

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ALTHOUGH MORE than a hundred years has elapsed since it was first described, Hodgkin's disease was clearer in the mind of the original describer than to anyone since his time. From his reports of cases it appeared that the duration of the disease was shorter and there was much less chance for the numerous clinical and pathologic variations to develop. Such diseases in the malignant lymphoma group as lymphosarcoma, leukemia, pseudoleukemia and mycosis fungoides are not only related to Hodgkin's disease in an unknown manner, but there may be a transition from one to the other members during the course of the disease.

Presented in this report are two cases of Hodgkin's disease, each having an unqualified microscopic diagnosis by many pathologists. Both patients have been observed for about 24 years. In one case there was a perplexing clinical diagnostic problem; in the other, therapeutic problems.

Since the customary attitude toward Hodgkin's disease has been one of pessimism insofar as prolongation of life is concerned, it is hoped that this report will be encouraging in that regard and will emphasize the value of persistent therapy. These cases further illustrate the extreme variability of the prognosis in this disease, due to factors of host resistance not yet understood.

REPORT OF A CASE

CASE 1. The patient, a married man, 30 years of age, was first observed in October, 1934. The primary complaint was intermittent rectal bleeding for two years, with soreness and some slight pain on defecation during the previous three months. Upon examination a papillary and ulcerated growth 3 cm. in diameter was noted on the left lateral wall of the ampulla of the rectum. No other abnormality was noted in a complete examination. The pathologist's report on a biopsy specimen was "Adenocarcinoma, Grade IV." Several surgeons said the lesion was inoperable. X-ray therapy was given at California Institute of Technology from September 27 to November 15, 1934; 3,600 r, measured in air, was delivered over each of the four pelvic portals. A recurrence of the growth appeared at the primary site in June, 1935, and radon seeds were implanted. In the following month the patient complained of shortness of breath and headache. There was pitting edema over the scalp, and pronounced swelling of the face and neck, with small nodes in the right and left supraclavicular fossae. Consideration was given to the possibility of lymphoma, particularly Hodgkin's disease, but the diagnosis decided upon was metastasis from the rectal carcinoma. X-ray therapy was given—2,000 r, measured in air, delivered to the three involved areas (supraclavicular fossae and mediastinum). In 1938, a shadow was demonstrated along the lateral border of the heart and this area received 2,000 r. In 1943, the patient com-

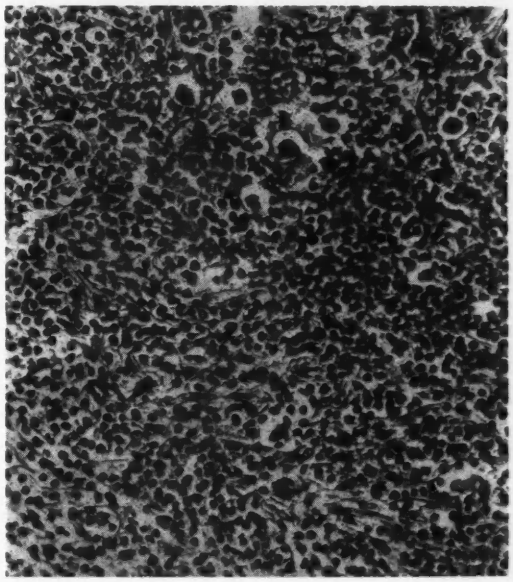

Figure 1 (Case 1).—Section of upper left abdominal mass showing Hodgkin's disease. Pathognomonic reticuloendothelial cells, including characteristic binucleate Reed-Sternberg cells are present.

Submitted July 10, 1958.

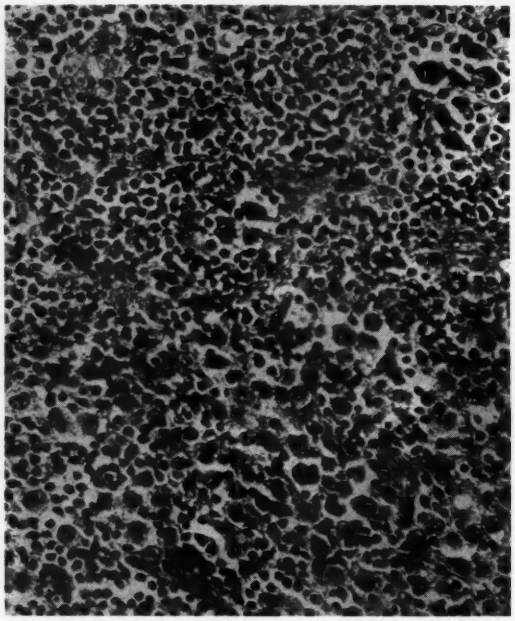

Figure 2 (Case 2).—Section of axillary lymph node showing Hodgkin's disease. Many large pathognomonic reticuloendothelial cells, including some binucleate Reed-Sternberg cells, are present. Characteristic nuclear chromatin arrangement is evident and prominent nucleoli are seen.

plained of an aching soreness in the epigastrium. The left lobe of the liver and spleen were palpable and the left hemidiaphragm was immobile. To determine the full extent of the disease and to obtain a biopsy specimen, laparotomy was done. The disease was confined to the epigastrium and involved lymph nodes, spleen and diaphragm. A positive microscopic diagnosis of Hodgkin's disease was finally established. Roentgen therapy again was given—2,000 r, measured in air, delivered through anterior and posterior portals to the left upper abdomen and diaphragm. For 15 years thereafter, up to the time of this report, the patient remained in excellent general condition and there was no recurrence.

Pathologist's Report. Upon microscopic examination of sections of the biopsy specimen, most of the tissue was found to be quite fibrous and portions were made of mature connective tissue lined by serosa. There was widespread proliferation of young fibroblasts in which there were scattered numerous endothelial cells with vesicular nuclei which varied somewhat in size and showed an occasional mitotic figure. A few cells had two or more nuclei and contained a moderate amount of cytoplasm; histologically they were Dorothy Reed cells. Lymphocytes were present in moderate number and in a few places there were occasional polymorphonuclear cells. In some portions there were more endothelial cells than fibroblasts, and in other parts the converse; but on the whole most of the growth showed considerable fibrosis, and a moderate amount of collagenous stroma was present. The diagnosis was Hodgkin's disease.

CASE 2. The patient, a man 33 years of age, was first observed in February, 1936. The primary complaint was swelling of the left lower portion of the neck, first noticed in 1934. Operation was performed in February, 1935, and a diagnosis of Hodgkin's disease was made. Postoperative x-ray therapy was given in March, 1935, and further x-ray therapy in January, 1936. When the patient was first examined by one of the authors, in February, 1936, a visible and palpable mass was presented in the left supraclavicular fossa. Nodes were palpable in both axillae, and a mediastinal mass 8 cm. in diameter was demonstrated by x-ray. In the next 40 days the patient received the following roentgen dosages, measured in air, directed to the mediastinal mass: Anterior, 900 r; right lateral, 650 r; left lateral, 1,100 r; posterior, 650 r. The right axilla received: Anterior, 700 r; lateral, 800 r. The left axilla received 900 r through a lateral portal.* This x-ray therapy was tolerated badly (as was all subsequent roentgen treatment throughout the entire course of the disease). In July, 1936, the left upper mediastinum had not returned to a normal contour, and lymph nodes were still palpable in both axillae. Therefore an additional 900 r, measured in air, was given over the mediastinum, as well as 900 r over each axilla. Two years passed and, in February, 1938, nodes were involved in the right axilla, for which 1,200 r, measured in air, was delivered. The patient then had a remission that lasted ten years, until September, 1948. Then recurrence developed in the mediastinum. Two cycles of x-ray therapy were given to the mediastinum during that year, one in May and the other in October. The total dosage of both series of treatments amounted to 3,600 r, measured in air, to the anterior mediastinum, 2,000 r to the right lateral, 2,400 r to the left lateral, and 2,400 to the posterior portal. In November, 1950, three nodes became enlarged in the right supraclavicular fossa and 2,000 r, measured in air, was delivered.

As the patient's mental outlook became quite pessimistic at this time, it was necessary to see him frequently for encouragement and to make sure he followed a high protein diet with dietary and vitamin supplements.

A year later a node became palpable in the right mid-neck and 2,000 r, measured in air, was delivered. The second longest remission, five years, lasted from 1951 to April 1956. Then a coalesced mass of small nodes appeared in the left lower neck and 2,000 r was delivered. The condition of the patient remained good, as it had since the onset of the disease.

In March, 1958, he had some pain over the dorsal spine. In an x-ray film of the chest evidence of a

*The same x-ray machine was used in both cases presented in this report. The factors were as follows: 200 KVP; 15 ma.; 50 cm. target-skin distance; thoreus filter, giving a half value layer of 1.9 mm. copper.

widening of the paraspinal soft tissue, and films of the spine confirmed this condition. Because of radiation changes in the skin, residual from the previous protracted irradiation, it was deemed advisable to treat this new lesion by a combination of nitrogen mustard and low dosage x-ray therapy. The patient was given 2.0 mg. of nitrogen mustard, then 1,500 r, measured in air, to the thoracic paraspinal mass, utilizing 400 KVP technique.

Pathologist's Report. In sections of lymph node some portions showed varying degrees of fibrosis. Parts of the fibrotic tissue were quite dense and contained very few cells. The entire architecture of the node was disrupted. There was pronounced proliferation of endothelial cells, some of which were multinucleated, forming Dorothy Reed cells. A number of polymorphonuclear leukocytes were mixed with the endothelial cells. A few eosinophiles were seen. The diagnosis was Hodgkin's disease.

Comment

The foregoing case illustrates the more common course of Hodgkin's disease and the problems of therapy. The patient was not as robust or as dynamic as the one described in Case 1. He never made a concerted effort to take the best of care of himself. During the 24-year period that he was under the authors' care, he had 202 examinations and 135 x-ray treatments.

DISCUSSION

X-ray Therapy

The best specific treatment to local areas of Hodgkin's disease is x-radiation. In certain cases in which minimal disease is confined apparently to one superficial node-bearing area, such as unilateral cervical involvement, the therapy of choice is irradiation carried to high dosage levels, with intent to cure. An alternative treatment form is radical surgical operation on the involved area. Heavy irradiation is also recommended if there is mediastinal involvement and for cases in which only two or three areas are clinically involved. When the disease is more generalized, therapy must be divided over all areas, but the dosages are still carried to moderate levels in order to lessen the chance of recurrence in the treated areas. It is well to emphasize two points: First, therapy will fail if it is given at the expense of the patient's general condition; second, the disease must be treated aggressively—a minimum of 2,000 r tumor dose in a relatively short time.

Surgical Treatment

In selected cases surgical operation is definitely indicated. When the disease is confined to one of the superficial node areas, such as the neck or groin, operation may be the treatment of choice. In cases in which the disease is unilateral and confined to the neck, radical neck dissection has been very successful. Several patients so treated have remained well for years without evidence of local recurrence. In a case observed by the authors, a radical groin dissection was performed on a 14-year-old girl in 1955, in lieu of x-ray therapy, and the patient had remained free of disease to the time of this report. However, Craver[1] advised surgical operation only in the exceptional case and recommended postoperative x-ray therapy in heavy dosages over the operative field.

Chemotherapeutic Agents

During the past 15 years, nitrogen mustard has proved a valuable aid for patients with generalized disease; and more recently it has been used to complement x-ray therapy in order that less radiation may be required. Nitrogen mustard may be used temporarily as a life-saving measure, because of its decongestant effect, by giving it before x-ray therapy where often congestant effects of the initial dose cause injurious results. There is no evidence that nitrogen mustard can bring about long remissions such as radiation produces. TEM (triethylenemelamine) is occasionally effective where nitrogen mustard is not, and has the advantage that it may be given orally. Hormone therapy, with corticotropin (ACTH), cortisone or prednisone, has on occasion caused partial regression of lesions in Hodgkin's disease. These are effective particularly in relieving itching, fever and pain, and to an extent, in combating anemia.

Radioactive isotopes have not proved of special benefit in the treatment of Hodgkin's disease.

Both of the patients reported upon herein had meticulous follow-up observations, which is essential in the management of this disease. There are two equally important aspects of treatment: (1) maintenance of the general physical condition, and (2) specific therapy for the involved areas.

The psychological approach to patients with Hodgkin's disease is much the same as that used in cases of tuberculosis—healthy respect for the disease and a hopeful attitude toward it. All supportive measures are advised—regular rest and exercise, a high protein diet, supplementary foods, vitamins, liver extracts, administration of hormones and transfusions to tide over the threatening episodes. Although it is well to practice forward-looking, aggressive treatment, the hope must be kept in mind that there will be more effective therapy in the future.

NOTE: The histologic slides of specimens from the two patients reported upon herein were reviewed by eight pathologists of the Senior Study Group at the Tumor Registry of the Cancer Commission of the California Medical Association. All made a diagnosis of Hodgkin's disease.

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Serious Human Infections Due to Bacilli of the Arizona Group

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ORIGINALLY ISOLATED from sick Gila monsters, horned lizards, and a chuckawalla lizard in 1939, the first discovered strain of the Arizona group of enteric bacilli was considered to be an unusual salmonella variant.[3,10] Then, in the next few years, members of the Arizona group of organisms were identified in material from reptiles, birds, domestic animals and man throughout the United States and in every continent.[5,8,13] There is now abundant evidence that strains of these motile Gram-negative bacilli infect a variety of hosts, commonly invading the blood and producing diseases with high mortality rates in young animals and birds. Particularly troublesome economically have been multiple outbreaks of bird septicemia and diarrhea in turkey farms in California and other states. Loss of as much as 60 per cent of a flock of young turkeys has been reported. These outbreaks have been carefully traced to commercial hatching eggs and to dried egg powder.[7]

Largely through the work of Edwards and his co-workers the Arizona group has been separated from the multitude of enteric bacilli and has been found to comprise a compact biochemical group of bacteria related to both coliforms and salmonellas.[4,6,7,13] They differ enough serologically and biochemically, however, to be regarded by authorities in this field as a separate individual genus, of which approximately 150 serotypes are now recognized.[5,11] Now that these organisms have been more clearly defined, it is no longer appropriate to place them in the vague category of "paracolons."

Infections in man are manifested by a variety of clinical forms, in general resembling salmonella infections. The cases recorded have been even somewhat more severe than the usual cases of salmonellosis, although more experience is needed to determine whether this apparent severity reflects factors of selectivity in recognizing Arizona infections. Mass infections from ingestion of food, with vomiting, diarrhea and fever, have occurred, in which Arizona strains were incriminated. Some of these outbreaks were traced to food such as ice cream and cream pie and to food handlers.[6,12] Typhoidal syndrome has been observed.

Certain serological strains appear to have a propensity for producing localized infections or abscesses. Strains of the commonest serotype in human infections (7:1, 2, 6) appear to be especially invasive. Over half of the strains of this serotype were isolated from infections of organs and tissues other than the intestinal tract in the series of Edwards, McWhorter and Fife.[6] The strains isolated in both of the cases reported herein are members of this serotype (7:1, 2, 6). This type is widely distributed in fowls in California.

Various localized human infections from which members of the Arizona group have been isolated include brain abscess, pleuritis, scalp abscess, conjunctivitis, osteomyelitis,[9] otitis media[2] and liver abscess.

Five deaths apparently resulting from infection by Arizona strains have been listed by Edwards, McWhorter and Fife[6]: (1) A 58-year-old man with severe gastroenteritis. (2) An undescribed patient with an hepatic abscess. (3) A 5-month-old infant with diarrhea. (4) A 5-month-old infant from which a member of the Arizona group was cultured from cardiac blood. (5) One of the cases (Case 1) reported herein.

In spite of the accumulating references to cases of serious human infection in public health reports and microbiological journals, few detailed clinical studies[8,9,12,14] and no detailed autopsy reports have been published.

In the two following cases the chief pathologic lesions consist respectively of (1) a fatal hepatic abscess, and (2) chronic osteomyelitis of the left femur with septic arthritis of the knee joint. In both cases bacilli of the Arizona group were isolated in pure culture from the lesions.

REPORT OF CASES

CASE 1. An 87-year-old white man, was admitted to Santa Clara County Hospital from a lodge rest home where he had been receiving domiciliary care. On the day before admission, convulsions developed unexpectedly. When a physician who was called noted that the temperature was 104°F. and that glycosuria was present, the patient was sent to Santa Clara County Hospital. Upon examination there, the patient was observed to be obese, senile, confused, lethargic and febrile. Abdominal guarding was noted. Neither jaundice nor diarrhea was present.

Leukocytes numbered 11,400 per cu. mm., of which 90 per cent were neutrophils. The blood sedimentation rate was 31 mm. in one hour (Westergren). Blood glucose was 330 mg. per 100 cc. Urinalysis showed 4 plus glycosuria and 2 plus acetonuria. A culture of blood grew no organisms. An area of patchy density in the base of the left lung was observed in an x-ray film of the chest.

The hyperglycemia and mild acetonuria were easily controlled by intravenous fluids and relatively small doses of regular insulin. However, the patient remained stuporous and confused. A spinal puncture was done the day after admission and the initial pressure was 200 mm. of water. The spinal fluid was clear and colorless and contained 4 leukocytes per cu. mm. Cultures of spinal fluid grew no organisms. On the day following admission, the serum sodium was 130 mEq. per liter, the potassium was 5.1 mEq./l. and the CO_2 was 29 mEq./l. The tem-

From the Santa Clara County Hospital, San Jose.

Presented in part before the Section on Pathology and Bacteriology at the 87th Annual Session of the California Medical Association, Los Angeles, April 27-30, 1958.

perature remained elevated, going as high as 104.4°F. (rectal). Hiccupping was an almost continual problem. The patient grew weaker without improvement and died four days after admission. Therapy from the time of admission consisted of penicillin (600,000 units intramuscularly daily), streptomycin (0.5 gm. initially and 0.5 gm. every 6 hours intramuscularly), digoxin (0.5 mg. daily), continual administration of oxygen nasally, and fluids intravenously and subcutaneously.

At autopsy five hours after death, on gross examination three apparently healing, superficial ulcers were noted on the left posterior trunk and there was a similar ulcer over each lateral malleolus. The liver weighed 2030 gm. and was reddish-brown, smooth and glistening. On the superior surface over the right lobe adjacent to the diaphragm was an irregular yellowish area of softening, 10 cm. across. Beneath this area, on sectioning, a large irregular abscess up to 12 cm. was exposed (Figure 1). The abscess was poorly demarcated, multiloculated and filled with yellow-gray viscid purulent material, with several indefinite smaller necrotic foci surrounding the abscess in a patchy distribution. The remainder of the liver substance was uniformly reddish-brown. The gallbladder was scarred and contracted about a single large 3 cm. calculus. No evidence of acute inflammatory reaction was noted in the gallbladder or biliary ducts. The lungs were hyperemic and showed evidence of patchy bronchopneumonia. The heart was moderately concentrically enlarged to 500 gm. The myocardium showed no changes, and there was moderate (Grade II-III) atherosclerosis of coronary arteries. The valves showed no important changes. The spleen was enlarged to 320 gm. and it was soft and mushy, the cut surfaces bulging. The pancreas appeared normal. No significant changes were observed in the entire gastrointestinal tract. Except for a moderate degree of cortical scarring no abnormality was seen in the kidneys. Upon microscopic examination of the liver a multiloculated abscess composed of necrotic hepatic cells and polymorphonuclear exudate was observed. Patchy collections of polymorphonuclear cells were scattered throughout the adjacent hepatic parenchyma (Figure 2). Gram-negative bacilli were easily seen in the abscess. Culture of material from the abscess grew bacilli of the Arizona group. Foci of acute bronchopneumonic exudate were present in the lungs. The splenic red pulp was hypercellular and contained numerous polymorphonuclear and plasma cells. The gallbladder wall was thickened and scarred, without evidence of acute inflammation. Sections of both kidneys showed moderate arterial and arteriolar hypertensive changes. Distinct microscopic stigmata of diabetes mellitus as described by Bell[1] were not found in the kidney or pancreas.

No clinical or autopsy evidence was found to suggest the manner of development of the liver abscess. It seems unlikely that the contracted gallbladder with its calculus was a primary portal of entry. The small skin ulcers also appear unlikely.

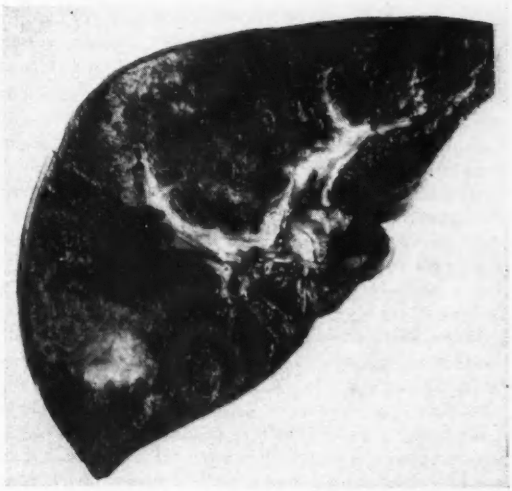

Figure 1 (Case 1).—Gross view of liver, showing irregular multiloculated abscess in right lobe containing yellow-gray viscid purulent material. Several indefinite smaller necrotic foci surround the abscess in a patchy distribution.

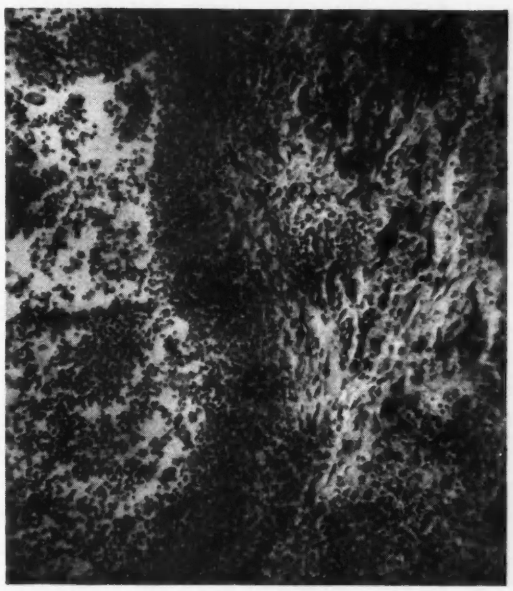

Figure 2 (Case 1).—Microscopic section of liver showing abscess containing acute polymorphonuclear exudate and adjacent necrotic hepatic parenchyma. (Magnification ×80).

No bacteriologic investigation was made of the environment from which the patient was admitted, but it appears most likely that the infection was acquired from contaminated food. The significance of hyperglycemia and glycosuria in this case is conjectural. It may be stated that pyogenic abscesses are not a common complication of diabetes mellitus. From the other point of view, diabetes mellitus has not

been described as occurring in any of the other case reports of Arizona infections.

CASE 2. A 63-year-old white housewife was sent by a physician to Santa Clara County Hospital September 2, 1958, because of fever and painful swelling of the left knee. For the previous ten months she had noted gradually increasing aching and swelling of the left knee, which was aggravated by walking. She recalled no significant trauma to the knee. One week before admission the referring physician had aspirated some fluid from the knee joint. The swelling rapidly recurred. Serious past illnesses included acute thrombocytopenic purpura in 1947, for which splenectomy was done, apparently with cure. In 1950 jaundice developed for a short time. This was apparently due to viral hepatitis, and there were no sequelae.

Upon examination at this hospital the chief physical findings were temperature of 103°F. and a swollen, tender and fluctuant left knee. The hemoglobin was 9.2 gm. per 100 cc. and the hematocrit 32 per cent. Leukocytes numbered 16,950 per cu. mm., of which 78 per cent were neutrophils. The urine was normal except for a trace of albumin. The serum albumin was 3.3 gm. per 100 cc., the globulin 3.1 gm. per 100 cc. and the serum total bilirubin 0.9 mg. per 100 cc. The serum VDRL was reactive. Several abnormalities were observed in the left femur in x-ray films taken at the time of admission. These consisted of an irregular sclerosis at the margins of the lower third of the shaft of the femur with periosteal proliferation. Several oval radiolucent areas within the shaft of the femur suggested localized bone absorption. There was no break in the cortex. The appearance was that of chronic osteomyelitis involving the lower third of the femur and its periosteum.

On the day the patient was admitted the knee was aspirated and 450 cc. of thick fluid resembling tomato soup was withdrawn. Cultures of this fluid grew bacilli of the Arizona group. On the sixth day after admission the focus of osteomyelitis was curetted through a 2.5 x 1.5 cm. window in the cortex of the lateral aspect of the distal femur. The cortex had a rough chalky appearance. A copious amount of whitish-yellow cheesy material was curetted from the marrow cavity. No pus was noted in the marrow cavity. Microscopic sections of tissue taken from the knee joint showed nonspecific chronic inflammatory granulation tissue of no distinctive pattern.

Chemotherapy consisted of achromycin orally, 250 mg. four times a day for a prolonged period. The patient had a spiking fever at first, the temperature going up to 103°F. The temperature gradually subsided in the ensuing three weeks and then remained within a normal range. The surgical wound continued to drain fluid, however, and no improvement of the bony lesions was seen radiographically. A more extensive curettage of the medullary canal was performed December 3, 1958. Multiple cultures of curetted material grew Arizona bacilli of the strain previously isolated from the knee joint effusion.*

BACTERIOLOGIC OBSERVATIONS

Bacilli of the Arizona group were isolated in pure culture from infected material in both cases. In Case 1 direct smears of pus from the liver abscess taken at autopsy showed Gram-negative bacilli similar to coliforms. Pus was inoculated on eosin-methylene-blue agar, Salmonella-Shigella agar and nutrient blood agar plates, and small colorless colonies grew overnight on all plates. Cultures of these were subsequently identified as members of the Arizona group by Dr. P. R. Edwards at the Communicable Disease Center at Chamblee, Georgia. In Case 2 bacilli of the Arizona group were repeatedly isolated in pure culture from fluid aspirated from the left knee joint and from infected necrotic material curetted from the medullary canal of the left femur. Cultures of these organisms were also identified by Edwards. In both cases the strains were found to be members of the same serologic type (7:1, 2, 6).

Subcultures of both strains had the following fermentation reactions: Lactose, no fermentation at 24 hours. (Acid was produced by subcultures of the strain of Case 1 after several transfers); salicin, no fermentation; sucrose, no fermentation; maltose, acid and gas; mannitol, acid and gas; dextrose, acid and gas; triple sugar iron agar, alkaline slant with acid, gas and H_2S in the butt. The organisms were motile, were negative for indole production and had no hydrolytic effect on urea. They caused a slight liquefaction of gelatin after ten days. There was positive reaction to methyl red but not to a Voges-Proskauer test.

Following the lead of Murphy and Morris,[12] who found that their infected patients developed agglutinating antibodies against the infecting Arizona strain, we tested the serum of the patient of Case 2 for the presence of antibodies against both Arizona strains which had been isolated in Case 1 and Case 2. Her serum was found to agglutinate Arizona bacilli isolated from her leg lesion to a serum dilution of 1:1280. In addition, serum from that patient agglutinated bacilli of the strain which had been isolated from the liver abscess in Case 1 to a serum dilution of 1:640. As a check against nonspecific cross reaction, serum of the patient in Case 2 was tested for antibodies against S. typhosa (H and O), S. paratyphi A, S. paratyphi B, and P. tularensis, and no agglutination was demonstrated against these.

DISCUSSION

The emergence of a newly recognized group of pathogenic enteric bacilli introduces several problems. It is apparent that bacterial pathogenicity is a relative quality, ranging at one extreme from the ability to develop progressive invasion of the body

*The patient died on February 6, 1959, 30 hours after another extensive sequestrectomy of the left femur. At autopsy there was bilateral bronchopneumonia. Organisms of the Arizona group were isolated at autopsy from the medullary cavity of the left femur but not from the lungs or blood.

when introduced by any route and at the other extreme to only a weak ability to multiply and grow when introduced directly into tissues. The ability of a given strain of enteric bacilli to produce symptoms in most of the individuals infected varies decidedly in both the species of hosts infected and the individuals infected. The evidence that members of the Arizona group are primary incitants of disease when ingested is very strong. The epidemiology of the infections in fowls has been particularly clear-cut, and there have been repeated instances in which it was possible to trace the transmission of infection through eggs.[5,7] Multiple outbreaks of severe gastroenteritis in man in which epidemiologically significant Arizona strains were isolated from the affected persons and not from asymptomatic persons have been well documented. Following the infections, the patients developed antibodies against the corresponding Arizona strain.[12] In some of these outbreaks the corresponding Arizona strain was isolated from food or food handlers. As a corollary to this, Arizona strains are rarely isolated from asymptomatic persons. Direct evidence of their ability to invade and multiply is supplied by the isolation of organisms from primary abscesses or other localized infections unassociated with the presence of foreign material in tissue and unassociated with other known pathogenic organisms.

Clinicians may well wonder why a group of organisms related to salmonellas and similar in their pathogenicity is not simply classified as a salmonella. The viewpoint of experienced workers in the isolation and epidemiology of enteric organisms is stated by Edwards, Cherry and Bruner[4]: "Without clear concise generic definitions [of salmonellas] the worker in a diagnostic laboratory is at a decided disadvantage. To relax the generic definition would lead inevitably to placing many atypical coliforms of doubtful pathogenic significance in that genus." For these reasons the Arizona species were placed in a separate genus.

Although they usually grow well on ordinary laboratory media used to isolate enteric pathogens, members of the Arizona group are difficult to recognize in the clinical laboratory and can be identified only by complex serological typing in special salmonella typing centers. In the clinical laboratory they are most likely to be confused with salmonellas, an error which is not especially serious. A much more important problem of recognition is encountered when an unrecognized Arizona strain ferments lactose early—and many strains apparently have this property.[15] The reason for this is that it is almost universal practice in clinical laboratories to consider a culture of an enteric bacillus which ferments lactose to be a nonpathogenic organism and to report it to the clinician as a coliform or paracolon. This reliance on lactose fermentation to screen out potentially dangerous organisms is, of course, very useful in general diagnostic bacteriological practice, but permits an unknown number of lactose-fermenting Arizona strains, as well as epidemiologically significant strains of E. coli, to go unrecognized.

An improved method for early recognition of this group in the clinical laboratory is needed.

SUMMARY

Bacilli of the Arizona group were isolated in pure culture of material from a fatal case of acute liver abscess in an 87-year-old white man and from a 63-year-old white woman who had chronic osteomyelitis of the left femur with septic arthritis of the knee joint.

The Arizona genus comprises a group of widely distributed pathogenic Gram-negative enteric bacilli which are related to both salmonellas and coliforms. Arizona strains have produced serious human infections with a high degree of invasiveness and occasional death. Members of this group are easily isolated and grown in the clinical laboratory, but are difficult to recognize.

ACKNOWLEDGMENT: The authors are grateful to Mrs. Elizabeth Smith, Librarian of Santa Clara County Hospital Library, for assistance in gathering the bibliography and to Messrs. Edward Chong and Charles Taddo of Santa Clara County Photography Department for assistance in preparing the illustrations.

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Q Fever Manifested by Anemia and Hepatitis

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Q FEVER is often thought of as a disease primarily involving the respiratory system and characterized clinically by patchy pneumonitis demonstrable by x-ray with associated fever, headache and myalgia. Actually, since Q fever is a systemic disease, its manifestations depend on the system or systems affected and do not necessarily include pulmonary involvement. Derrick, who first described Q fever in Australia in 1937,[6] noted respiratory symptoms consisting of slight cough in only two of nine patients. In one case the clinical manifestations consisted chiefly of anemia, jaundice and prolonged fever. Since that time Q fever has been reported from many areas in the world,[14] and cases with such variable manifestations as arthritis,[12] meningitis[8] and pericarditis[10] have been described.

The purpose of this report is to emphasize the systemic nature of Q fever and to illustrate the diagnostic problems in a case in which the striking clinical features were hepatitis, anemia and the absence of pulmonary involvement.

REPORT OF A CASE

A 50-year-old white man was admitted to hospital on May 3, 1958, with a diagnosis of fever of unknown origin of one month's duration.* He had first become ill on April 2, with a sudden attack of fever, myalgia, anorexia, left occipital headache and numbness along the distribution of the right ulnar nerve. There were no symptoms referrable to the respiratory tract. On subsequent physical examination no abnormalities were observed.

The patient said that he had been employed as a rural mail carrier in Lodi, California, for 17 years. Although he had made frequent visits to his brother's cattle farm, he denied direct contact with animals or ingestion of raw milk. He had no history of serious illnesses.

Treatment with tetracycline, 1 gm. daily for three days, and chloromycetin, 1 gm. daily for an additional three days, both given orally, was ineffective. The patient continued to have spiking fevers, with rises in temperature as high as 103°F. twice daily. Two weeks after the beginning of the attack, the hematocrit determination was 40 per cent. Liver function tests showed that bromsulfalein retention, albumin-globulin ratio and icteric index were within normal limits; however, cephalin flocculation was 4 plus in 24 hours. An x-ray film of the chest showed no abnormalities. Results of blood and urine cultures and agglutination tests for typhoid, paratyphoid and brucellosis were negative. Blood smears were negative for malaria. Serum drawn on April 18 and May 1 was sent to the laboratory of the California State Department of Public Health for viral and rickettsial studies.

When he entered the hospital on May 3, the patient had a temperature of 100°F. During the preceding month he had lost 17 pounds in weight. The liver had a firm edge but was not enlarged or tender. The lungs were clear to auscultation. There was no evidence of lymphadenopathy, splenomegaly or jaundice. Leukocytes numbered 8,000 per cu. mm.; the differential count was normal. Hematocrit determination was 44 per cent. Results of urinalysis were within normal limits. X-ray examination of the chest showed no abnormalities. Results of the following procedures were negative: Skin tests for tuberculosis, coccidioidomycosis and histoplasmosis, serologic tests for typhoid, paratyphoid A and B, brucellosis, tularemia and leptospirosis, a heterophil agglutination test, and repeated routine blood and urine cultures. No pathogenic bacteria or parasites were found in the feces. Results of liver function tests were as follows: Cephalin flocculation, 4 plus in 24 and 48 hours; thymol turbidity, 8 units; albumin, 3.1 gm. per 100 cc.; globulin, 4.6 gm. per 100 cc.; total bilirubin, 0.4 mg. per 100 cc., cholesterol, 203 mg. per 100 cc.; cholesterol esters, 143 mg. per 100 cc.; alkaline phosphatase, 6 units (Bodansky); bromsulfalein retention, 6 per cent in 45 minutes; prothrombin time, 90 per cent. Paper electrophoresis showed an increase in serum globulin content, primarily of the alpha-2 fraction. On biopsy, the liver showed no pathologic changes.

Spiking fevers continued for the next week. During this time a normochromic, normocytic anemia developed and the hematocrit concentration gradually dropped to a low of 28 per cent. Reticulocyte counts and bone marrow smears were normal. Results of a Coombs test and of repeated examinations of stool specimens for occult blood were negative. Urinary and fecal urobilinogen content were within normal limits. Blood volume studies utilizing radiochromium-tagged red cells showed a 20 per cent reduction in red cell mass, and frequent sampling indicated that the red cell survival time was normal.

A diagnosis was finally established by the demonstration of a rise in the titer of complement-fixing antibodies for Q fever from 1:16 in serum taken April 18 to 1:64 in serum taken May 1. The titer remained at 1:64 as determined by subsequent tests on May 12 and May 22. Although no specific therapy was given, the fever gradually subsided after a total duration of five weeks. On June 18, two and a half months after the onset of illness, the patient still complained of weakness and had occasional elevations in temperature to 99.6°F. (oral). The hematocrit concentration at that time had risen to 45 per cent, but the cephalin flocculation was 2 plus in 24 hours and 3 plus in 48 hours.

From the Department of Medicine, University of California School of Medicine.

Submitted August 29, 1958.

*Referred by H. E. Hoff, M.D., Lodi, California.

DISCUSSION

The diagnosis in the present case was established by the demonstration in the patient's serum of complement-fixing antibodies specific for Q fever.[1] The rise in antibody titer and a clinical course similar to that described in cases of Q fever in older adults[2] indicate that the infection was of recent origin. It is possible that an even greater rise in titer might have been observed if serum had been drawn earlier in the acute stage of the patient's illness. The fact that all other clinically compatible conditions were ruled out further supported the diagnosis. Also, the onset of the disease in April is in accord with the reported seasonal incidence of Q fever in Northern California.[5] Although the source of the infection could not be determined definitely, the patient's occupation as rural mail carrier and his exposure to livestock suggest inhalation of dust infected by animal excreta.

The principal clinical features were hepatitis, anemia, prolonged fever and the absence of pulmonary involvement. In such cases the differential diagnosis may appear to lie chiefly between infectious hepatitis, amebic hepatitis and the hepatitis of infectious mononucleosis.[2] In the present case the hepatitis was manifested by a firm liver edge, reversed albumin-globulin ratio and abnormal cephalin flocculation values. The latter was the striking and persistent finding; results of other liver function tests were either borderline or normal. Biopsy showed no pathologic changes in the liver, although Gerstl, Movitt, and Skahen[9] found that often in Q fever there is evidence of focal hepatocellular damage in liver biopsy specimens. They also commented on the lack of correlation between results of liver function tests and biopsy findings in Q fever, as in the present case.

The cause of the anemia which developed late in the course of the illness could not be determined. Since laboratory tests failed to show evidence of hemolysis or blood loss, the anemia may have been related to a decreased production of red blood cells. Anemia of uncertain cause may develop in any prolonged case of febrile rickettsial infection and a decreased red cell volume has been documented in epidemic typhus.[13] In a report of a fatal case of Q fever, Perrin[11] described areas of hypoplasia in specimens of sternal marrow taken at autopsy, although before death the hemoglobin content of the blood had been normal. Also of interest is the recent report of an "angiitis" in histologic sections of bone marrow from a patient with Q fever.[7] Clark and associates,[4] in an analysis of the clinical features in 180 cases of Q fever, noted a correlation between anemia and evidence of hepatic disease; conversely, anemia has been reported in patients without hepatitis.[12] In the case herein reported, anemia developed and subsequently disappeared without apparent relation to the hepatitis.

A febrile course of more than four weeks was described in 30 of the 180 cases of Q fever reported by Clark and associates.[4] In 28 of the 30 cases the patients were over 30 years of age, and several patients in this age group did not respond well to treatment with adequate doses of chlortetracycline.[3] In the present case the antibiotic therapy was not adequate in dosage or duration, but even without the use of antibiotics in the large doses usually required, the fever gradually subsided and the patient was well.

SUMMARY

Involvement of the respiratory tract is relatively frequent in Q fever. Since Q fever is a systemic infection, however, the clinical manifestations vary widely and do not always include pulmonary symptoms. This is particularly true in adults over 30 years of age. To illustrate the diagnostic problems in such instances, this report describes a case of Q fever characterized clinically by nonicteric hepatitis, anemia, prolonged fever and lack of clinical or x-ray evidence of pneumonitis.

University of California Medical Center, San Francisco 22 (Hurwitz).

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California MEDICINE

For information on preparation of manuscript, see advertising page 2

EDITORIAL

Twenty Years After

TWENTY YEARS AGO last month a group of serious and far-sighted California physicians brought California Physicians' Service into the world as the first statewide medically-sponsored medical care prepayment plan in the country.

Since that time C.P.S. has often been referred to as the "doctors' own baby." If this appellation is accurate, it is obvious today that the baby has grown to considerable stature while passing through its childhood, adolescence and teen-age periods.

Designed to provide a budget method for people of low incomes to meet their medical care costs, C.P.S. has served many additional purposes in its twenty years of existence.

Behind the whole concept—this is a bit of history which is new to many of California's more recent physicians—lay more than twenty years of study by physicians and others who were aware of the need of the common man to be able to budget his own funds so that he would be able to pay the physician and the hospital when these services were needed. The records behind C.P.S. go back to studies made as early as 1917.

Other studies, including the review of the costs of medical care under a voluntarily-financed commission headed by the late Ray Lyman Wilbur, the California medical-economic survey of 1934-1935, and the experience of several county medical societies in offering prepayment plans in other states, were added to the research material which went into California Physicians' Service.

An added impetus, probably not needed but certainly most suggestive, was the attempt of former California Governor Culbert L. Olson to have enacted a statewide compulsory health insurance plan as a part of the laws of California. Some observers claim that C.P.S. was created simply as the profession's answer to this threat; more knowledgeable and experienced physicians admit that the Olson threat served as a spur to the profession but was not the primary factor in the establishment of this new plan.

Regardless of the impact of political machinations on its origin, C.P.S. was incorporated in February, 1939, and started on an admittedly empirical course of providing complete medical and surgical services for subscribers at a low monthly rate of dues. The empirical part of the procedure was bound up in the fact that nobody had ever before tried to provide such a service; the utilization and cost of such service was based on nothing more than guesswork.

Within a short time the "bugs" in this approach began to show up. There was excessive utilization. (An extreme example: One patient visited five physicians on her day off from work.) There was an evident backlog of unmet medical needs (one family presented five children for simultaneous tonsillectomy soon after coverage was in effect). There were even excesses by some physicians who rationalized that the way to offset the low fees paid by C.P.S. on the basis of funds available to meet the units of medical service provided was to add more units to cover unneeded visits or even visits never made.

Some of these drawbacks were serious enough to cause entire county medical societies to doubt the wisdom of the program. In at least two instances, county societies threatened to "secede" from the California Medical Association.

With the advent of World War II came other problems. New citizens imported from other states were jammed into government housing projects, where their only access to medical care was through an organized program under which C.P.S. underwrote the cost of the service in exchange for a few dollars a month collected as part of the rent by the housing authorities. These programs, each organized around a single housing project, operated with varying degrees of dissatisfaction. Where the dues for housing project residents had been calculated

on the basis of normal expectations of illness, the actuaries had not calculated that the birth rate would jump from 22 births per thousand to more than 110. When these programs were finally dropped (after the war) C.P.S. could truthfully state that the only epidemic it had encountered in the war housing projects was an epidemic of babies.

Following the war, C.P.S. was called upon to act in behalf of the physicians in the home-town medical care program of the Veterans' Administration. This plan has now operated for more than 12 years and has been effective in providing veterans with home-town medical care for service-connected disabilities, rather than forcing them to travel to VA facilities and register as in-patients before they could see a physician.

More recently, C.P.S. has been asked to serve as fiscal agent for the "Medicare" program, serving the dependents of military personnel. In this role the organization has processed thousands of cases in which patients are treated by California physicians instead of being shunted to military hospitals staffed by drafted physicians.

In the past two years C.P.S. has again been asked to serve as a fiscal agent, this time under the social welfare laws. Today the organization is handling this work for 34 of the state's 58 counties.

These activities, coming on top of C.P.S.'s continued coverage of employed groups and individuals under the voluntary plans developed from the original prepayment concept, have demonstrated the versatility that C.P.S. can offer. Where many physicians in years past thought of C.P.S. as an interloping third party, they have now come to the conclusion that this is their own organization, their own shield against unwarranted interference in medical practice by government or commercial interests.

Today, after twenty years of growth, C.P.S. can proudly point to a record of more than a quarter billion dollars disbursed to the physicians of California in behalf of their enrolled patients. It can point to an annual business of more than $35 million in its regular program, plus added millions as agent for the Veterans' Administration, Medicare and the social welfare plans. It can, with notable justification, display itself as the one medical prepayment plan which, acting for, through and by physicians, protects the interests of all physicians and serves as a model for its contemporaries. Without C.P.S. in the picture, only wild conjecture could guess what progress would have been made in commercial and other prepayment plans operating without this constant spur and example.

California Physicians' Service has earned the respect and admiration of California physicians through the sheer weight of its positive accomplishments. It is fitting to salute it on the occasion of its completion of 20 years of service.

California MEDICAL ASSOCIATION

NOTICES & REPORTS

PAUL D. FOSTER, M.D.

Paul D. Foster, M.D., New C.M.A. President-Elect

NEW PRESIDENT-ELECT of the California Medical Association is Paul D. Foster, M.D., of Los Angeles.

Doctor Foster was elected at the C.M.A.'s 88th Annual Session held February 22 to 25 in San Francisco, succeeding T. Eric Reynolds, M.D., of Oakland. He will be installed as President at the C.M.A.'s annual meeting in 1960.

Doctor Foster has practiced medicine in California since 1935, specializing in dermatology and syphilology. He has wide experience in medical affairs on the local, state and national levels.

Vice-Speaker of the C.M.A. House of Delegates in 1955, he was, until elected to his new post, a Councilor on the state Association's Council, representing the Third District (Los Angeles County). He is a delegate to the American Medical Association and a member of the C.M.A. Committee on Postgraduate Activities, on which he serves as chairman of the Motion Picture Division. He is also a member of the Bureau of Economic Research and Planning of the C.M.A.

Doctor Foster has long been prominent in the activities of the Los Angeles County Medical Association, having served that organization with outstanding distinction as a councilor from 1945 to 1951; as secretary-treasurer for two consecutive terms, 1951 and 1952; and as president in 1953. He has been a member of the L.A.C.M.A. board of trustees since 1951.

Born at Corry, Pennsylvania, Doctor Foster received his preliminary education at Manual Arts High School and the University of California at Los Angeles.

He was graduated from the College of Medical Evangelists School of Medicine in Los Angeles in 1932. After serving his internship at Los Angeles County General Hospital, he received further training at New York Skin and Cancer Hospital and Clinic and also the New York Postgraduate Medical

School Department of Dermatology. He is a founder of the Student Medical Association.

A Diplomate of the American Board of Dermatology, Doctor Foster is an associate clinical professor of dermatology and syphilology at the College of Medical Evangelists School of Medicine, and is a member of the advisory committee to C.M.E.'s Audiovisual Postgraduate Extension Program, having served as director of the program until January of this year. He is director of medical education for Encyclopaedia Britannica Films, Inc.

Doctor Foster is a member of numerous professional, scientific and academic organizations, and the author of more than 50 scientific publications in his field. He is active in civic, fraternal and veterans affairs.

Doctor and Mrs. Foster reside at 2477 Ridgeway Road, San Marino, with their son and daughter, Lawrence Dean, 21, and Sandra Lee, 20. Both children are pre-medical students, Lawrence at U.C.L.A. and Sandra at the University of Arizona.

Biggest C.M.A. Meeting

New record highs in attendance of physicians and of guests were set at the eighty-eighth annual meeting of the California Medical Association which was held in San Francisco, February 22 to 25. Registration of physicians totaled 3,322. Guests—members of the Woman's Auxiliary, exhibitors, friends and members of the families of physicians—numbered 2,030. The total was 5,352.

Among its actions the House of Delegates:

Installed Dr. T. Eric Reynolds of Oakland as president.

Passed a resolution directing California Physician's Service to set up a prepayment medical care plan at reduced rates for persons over 65 years of age whose income is inadequate for full payment.

Adopted a committee recommendation that employers be urged to abandon arbitrary retirement of workers at age 65.

Set dues for the 1959-1960 fiscal year at $55 per member.

Elected the following California Medical Association officers and councilors and American Medical Association delegates and alternates. (An additional councilor was elected to represent District 7 owing to an increase in the membership of San Francisco Medical Society to more than one thousand; and an additional delegate to the A.M.A. was elected, bringing California's total to 17, as a result of the increase in C.M.A. membership to more than 17,000 during the past year.)

In the following lists an asterisk (*) beside a name indicates the person elected was the incumbent.

California Medical Association officers elected:

President-Elect.....Paul D. Foster, Los Angeles
Speaker of the House of Delegates.....James C. Doyle,* Los Angeles
Vice-Speaker.....Ivan C. Heron,* San Francisco

* * *

Councilors, elected for three-year terms:

Omer W. Wheeler,* Riverside. *Second district*—Imperial, Inyo, Mono, Orange, Riverside and San Bernardino counties.

Malcolm C. Todd,* Long Beach. *Third district, Office No. 1*—Los Angeles county.

Arthur F. Kirchner,* Los Angeles. *Third district, Office No. 4.*

William F. Quinn, Los Angeles. *Third district, Office No. 2* (to serve out unexpired term of Paul D. Foster, President-Elect).

Byron L. Gifford, Santa Barbara. *Fourth district*—San Luis Obispo, Santa Barbara and Ventura counties.

Samuel R. Sherman,* San Francisco, and
Donald M. Campbell, San Francisco. *Seventh district*—San Francisco county. (Campbell elected to new seat created by increase in membership.)

Ralph C. Teall,* Sacramento. *Tenth district*—Alpine, Amador, Butte, Colusa, El Dorado, Glenn, Lassen, Modoc, Nevada, Placer, Plumas, Sacramento, Shasta, Sierra, Siskiyou, Sutter, Tehama, Trinity, Yolo and Yuba counties.

* * *

Election of delegates and alternates to A.M.A. for two-year terms:

Delegates	*Alternates*
Henry Gibbons, III* San Francisco	Claude P. Callaway* San Francisco
Sam J. McClendon* San Diego	John M. Rumsey* San Diego
Eugene F. Hoffman* Los Angeles	Gerald W. Shaw* Santa Monica
Warren L. Bostick San Rafael	Francis H. O'Neil Eureka
J. B. Price Santa Ana	Charles Hudson Oakland
Frank A. MacDonald* Sacramento	J. E. Vaughan* Bakersfield
Paul D. Foster* Los Angeles	Arthur A. Kirchner* Los Angeles
Donald A. Charnock* Los Angeles	Carl M. Hadley* San Bernardino
H. Milton Van Dyke Long Beach	Omer W. Wheeler Riverside
	Samuel R. Sherman† San Francisco

* * *

C.P.S. Trustees—Three-year term:

Dr. Bert Halter,* San Francisco; Dr. Paul I. Hoagland,* Pasadena; Mr. Guy W. Wadsworth, Jr.,* Los Angeles; Dr. Angus C. McDonald, Los Angeles; Dr. John E. Vaughan, Bakersfield.

* * *

In an organizational change, the House of Delegates amended the by-laws to permit the appointment of someone other than the secretary of the Association to the chairmanship of the Committee on Scientific Work. This was done in order that Albert C. Daniels, who has resigned as secretary, may continue his work with the committee. A new secretary has not yet been named.

†Takes place of H. Milton Van Dyke as alternate to J. Lafe Ludwig.

In Memoriam

AREHART, ARTHUR A. Died in Palo Alto, January 2, 1959, aged 63. Graduate of Long Island College Hospital, Brooklyn, New York. Licensed in California in 1927. Doctor Arehart was a retired member of the Monterey County Medical Society and the California Medical Association, and an associate member of the American Medical Association.

✤

BARRETT, GILBERT MICHAEL. Died January 10, 1959, aged 88. Graduate of Bellevue Hospital Medical College, New York, 1895. Licensed in California in 1898. Doctor Barrett was a member of the San Francisco Medical Society.

✤

BOST, FREDERIC CARROLL. Died in Belvedere, February 2, 1959, aged 58, of heart disease. Graduate of the University of California School of Medicine, Berkeley-San Francisco, 1926. Licensed in California in 1926. Doctor Bost was a member of the San Francisco Medical Society.

✤

CARY, RAYMOND JOHN. Died in Long Beach, January 7, 1959, aged 73. Graduate of Johns Hopkins University School of Medicine, Baltimore, Maryland, 1912. Licensed in California in 1918. Doctor Cary was a retired member of the Los Angeles County Medical Association and the California Medical Association, and an associate member of the American Medical Association.

✤

CHERWIN, NATHAN HALE. Died December 21, 1958, aged 59. Graduate of Columbia University College of Physicians and Surgeons, New York, 1924. Licensed in California in 1924. Doctor Cherwin was a member of the Los Angeles County Medical Association.

✤

COOLEY, CHESTER LYNN. Died January 22, 1959, aged 64, of heart disease. Graduate of Stanford University School of Medicine, Stanford-San Francisco, 1928. Licensed in California in 1928. Doctor Cooley was a member of the San Francisco Medical Society.

✤

DARLING, HERBERT HENRY. Died in San Rafael, January 23, 1959, aged 58. Graduate of Harvard Medical School, Boston, Massachusetts, 1925. Licensed in California in 1925. Doctor Darling was a member of the San Francisco Medical Society.

✤

DICKSON, ARCHIBALD JOHN. Died in Altadena, January 11, 1959, aged 79. Graduate of McGill University Faculty of Medicine, Montreal, Quebec, 1903. Licensed in California in 1932. Doctor Dickson was a retired member of the Los Angeles County Medical Association and the California Medical Association, and an associate member of the American Medical Association.

✤

FREYERMUTH, OTTO GEORGE. Died in San Francisco, January 19, 1959, aged 82. Graduate of the Hahnemann Medical College and Hospital, Chicago, Illinois, 1903. Licensed in California in 1903. Doctor Freyermuth was a member of the San Francisco Medical Society.

✤

HAIRE, ROBERT DONNELL, JR. Died in Glendale, January 12, 1959, aged 54. Graduate of Jefferson Medical College of Philadelphia, Pennsylvania, 1928. Licensed in California in 1952. Doctor Haire was a member of the Los Angeles County Medical Association.

JAMESON, JANE. Died in Castro Valley, January 20, 1959, aged 35. Graduate of University of Wisconsin Medical School, Madison, Wisconsin, 1948. Licensed in California in 1953. Doctor Jameson was a member of the Alameda-Contra Costa Medical Association.

✤

JONES, JOSEPH LAURENCE. Died in Los Angeles, January 5, 1959, aged 63. Graduate of Columbia University College of Physicians and Surgeons, New York, 1919. Licensed in California in 1943. Doctor Jones was a member of the Los Angeles County Medical Association.

✤

REIS, ABILIO G. DE SILVA. Died in Piedmont, February 3, 1959, aged 66. Graduate of Universidade de Lisboa Faculdade de Medicina, Portugal, 1919. Licensed in California in 1922. Doctor Reis was a member of the Alameda-Contra Costa Medical Association.

✤

SCHOONMAKER, GUY DANIEL. Died in San Francisco, January 14, 1959, aged 67, of heart disease. Graduate of Vanderbilt University School of Medicine, Nashville, Tennessee, 1917. Licensed in California in 1922. Doctor Schoonmaker was a member of the San Francisco Medical Society.

✤

SEKIYAMA, ISAMI. Died January 1959, aged 67. Graduate of Tohoku Imperial University Medical Faculty, Sendai, Japan, 1915. Licensed in California in 1919. Doctor Sekiyama was a member of the Los Angeles County Medical Association.

✤

STANIFORD, KENNETH J. Died in Fresno, January 25, 1959, aged 69. Graduate of Cooper Medical College, San Francisco, 1911. Licensed in California in 1911. Doctor Staniford was a retired member of the Fresno County Medical Society and the California Medical Association, and an associate member of the American Medical Association.

✤

TAYLOR, J. PHYROMM. Died in Los Angeles, December 29, 1958, aged 60, of heart disease. Graduate of Meharry Medical College, Nashville, Tennessee, 1922. Licensed in California in 1936. Doctor Taylor was a member of the Los Angeles County Medical Association.

✤

TUOHY, EDWARD BOYCE. Died in Los Angeles, January 12, 1959, aged 50, of a cerebral hemorrhage. Graduate of University of Pennsylvania School of Medicine, Philadelphia, 1932. Licensed in California in 1939. Doctor Tuohy was a member of the Los Angeles County Medical Association.

✤

VAN PELT, CLIFFORD A., JR. Died January 27, 1959, aged 49. Graduate of University of Kansas School of Medicine, Lawrence-Kansas City, 1937. Licensed in California in 1946. Doctor Van Pelt was a member of the Los Angeles County Medical Association.

✤

WILSON, GUSTAVE. Died in Sacramento, December 3, 1958, aged 83, of complications of cerebral vascular disease. Graduate of University of Michigan Homeopathic Medical School, Ann Arbor, 1903. Licensed in California in 1904. Doctor Wilson was a member of the Sacramento Society for Medical Improvement.

NINTH ANNUAL
REGIONAL POSTGRADUATE INSTITUTE
SOUTHERN COUNTIES

Presented by Committee on Postgraduate Activities of the California Medical Association, in cooperation with Orange, Riverside and San Bernardino County Medical Societies, and the University of California, San Francisco, Seymour M. Farber, M.D., Assistant Dean for Continuing Medical Education.

Disneyland Hotel . . . April 23 and 24, 1959

PROGRAM

THURSDAY, APRIL 23, 1959

MORNING SESSION

Emotional Problems in the Daily Practice of Medicine

9:00-9:45 a.m.—**Emotional Problems of Patient and Doctor**—Edwin Frederick Alston, M.D.

9:45-10:30 a.m.—**The Psychotherapeutic Approach to Emotional Problems**—Edward M. Weinshel, M.D.

10:45-12:00 noon—**Three Panel Symposia** (you may go to one of your choice):
The Nervous Patient in Office Practice
The Psychotherapeutic Value of Listening
Psychological Impacts of Illness

12:00-2:00 p.m.—Luncheon
Panel: **Face to Face with the Psychiatrist**

AFTERNOON SESSION

Gastrointestinal Motility

2:00-2:45 p.m.—**The Medical Approach to Nausea, Vomiting and Heartburn**—John V. Carbone, M.D.

2:45-3:30 p.m.—**The Diagnosis and Treatment of Intestinal Gas, Pain and Diarrhea**—Emanuel Friedman, M.D.

3:45-5:00 p.m.—**Three Panel Symposia—Emotions and the Gastrointestinal Tract** (you may go to one of your choice):
Emotions and the Upper Gastrointestinal Tract
Emotions and the Middle Gastrointestinal Tract
Emotions and the Lower Gastrointestinal Tract

7:30-10:00 p.m.—Cocktails and Dinner (wives invited)

FRIDAY, APRIL 24

MORNING SESSION

Gynecologic Endocrinology

9:00-9:45 a.m.—**Endocrinologic Aspects of the Pubescent Period**—Paul Scholten, M.D.

9:45-10:30 a.m.—**Endocrinologic Procedures and Practices in the Menopausal Patient**—Edmund W. Overstreet, M.D.

10:45-12:00 noon—**Three Panel Symposia** (you may go to one of your choice):
Emotional Problems in Marriage
Emotional Problems in Pregnancy
Emotional Problems in Menopause

12:00-2:00 p.m.—Luncheon
Panel: **Psychological Resources of the Physician**

AFTERNOON SESSION

General Surgery

2:00-2:45 p.m.—**Surgical Considerations and Treatment of Pain**—Benson B. Roe, M.D.

2:45-3:30 p.m.—**Surgery in the Dying Patient**—Orville F. Grimes, M.D.

3:45-5:00 p.m.—**Three Panel Symposia** (you may go to one of your choice):
The Surgeon and Authority
Psychological Reactions of the Patient to Surgery
Emotional Recovery and Rehabilitation Following Surgical Procedures

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HOST: Orange County Medical Society . . . **REGIONAL CHAIRMAN:** Edmund F. Cain, M.D., 200 N. Palm, Anaheim, California . . . **Institute Fee:** $25.00 . . . For additional information contact Postgraduate Activities office, California Medical Association, 2975 Wilshire Boulevard, Los Angeles 5. **All California Medical Association members and their families are cordially invited to attend.**

PUBLIC HEALTH REPORT

MALCOLM H. MERRILL, *M.D., M.P.H.*
Director, California State Department of Public Health

THE California Tumor Registry received its 200,000th report of neoplasm during the first week in January, maintaining its standing as the largest single registry in the world.

The registry is now engaged in a series of studies based upon information in its files. One such study, a collaborative effort of the registry, the C.M.A. Cancer Commission and the California Society of Pathologists, will test the validity of published survival rates for cancer of the breast. Three outstanding pathologists in the state are independently rereading the slides on which the original diagnoses of breast cancer were based.

Decided improvement in quality of pathologic specimens has been recorded in the 15-year experience of cancer cases in the 38 hospitals currently participating in the registry. The proportion of cases confirmed by histopathology has risen from 73 per cent in 1942-46, to 92 per cent in 1956. Follow-up information is being obtained on 91 per cent of all cancer cases.

* * *

The department's Viral and Rickettsial Disease Laboratory is working on a simplified test for the diagnosis of rabies. Once perfected, the test will remove all doubt in a matter of days, making known whether the rabies vaccine treatment must be started.

At present, prompt diagnosis depends upon finding specific virus inclusion bodies in diseased tissues of the suspected animal. Sometimes these inclusions are absent or not typical, making quick microscopic diagnosis difficult or impossible.

For this reason, studies are underway to develop a simplified test. The laboratory is using a specific antibody to which a fluorescent dye is coupled chemically. If rabies virus is present in the tissue the antibody added unites with the virus. Since the antibody is tied to the fluorescent dye, this highly specific fluorescence can be seen through the microscope when ultraviolet light is shined upon the tissue.

* * *

The State Advisory Hospital Council held public hearings January 21 and 22 in Los Angeles to consider policies for the fiscal year beginning July 1, and to reallocate state and federal hospital construction funds of $1,776,818.

The council recommended establishment of planning objectives for hospital development in the Los Angeles metropolitan region, which includes all of Los Angeles and Orange counties, and the contiguous areas of Riverside and San Bernardino counties.

The planning is based on an anticipated population of 12,000,000 in the region by 1975, and consideration is being given to the geographic distribution of this population and the development of hospitals between now and 1975 in an orderly manner to meet the communities' needs at that time.

Metropolitan regional planning in the state has been endorsed and supported by the California Hospital Association, the Hospital Council of Southern California and the Welfare Planning Council of Los Angeles.

The need for such planning has been recognized and emphasized by the experience in hospital development during the last ten years. Between 1950 and 1958, 56 out of the 67 hospitals built in Los Angeles County were under 100 beds. If this pattern continues through 1975, there will be 204 hospitals under 100 beds out of a total of 290 hospitals in the county. The construction of hospitals large enough to provide total patient services is perhaps the most difficult problem in developing long-range planning for metropolitan regions.

The council recommended for the 1959-60 State Plan for Hospital Construction the establishment of the Los Angeles metropolitan region with long-range planning objectives and criteria for development of individual facilities within the region. Within the region, individual hospital service areas are established to delineate geographic areas which have some community of interest for hospital and medical purposes related to an established community center, and which may be expected to have an estimated minimum population of 250,000 by 1975.

In addition, individual general hospitals in each area should provide for a minimum capacity of 150 beds. The policies and criteria established for the Los Angeles metropolitan region for 1959-60 will be subject to annual review and modified as warranted by changing conditions. In addition,

basic policies developed for this region will be reviewed in connection with study of the other metropolitan regions in California.

During 1959-60, in the allocation of state and federal funds, general hospital projects in the Los Angeles metropolitan area shall not be considered unless they propose complete facilities of not less than 150 general hospital beds each. These policies will not in any way change the existing methods for estimating need for hospital beds in individual areas or in the determination of priority sequence for consideration of allocations.

From funds allocated August 12, 1958, $1,776,818 in state funds and an equal amount in federal funds were not utilized by three hospitals and were reallocated to the following projects: East Bay Lutheran Hospital, San Leandro, $663,894; St. Rose Hospital, Hayward, $181,128; San Jose Hospital, San Jose, $25,000; Rancho Los Amigos, Downey, $408,090; Santa Clara County Hospital, San Jose, $498,706; Fairmont Hospital of Alameda County, $268,430.

* * *

Two members of the department's Viral and Rickettsial Disease Laboratory have contributed chapters to the recently published third edition of the book "Viral and Rickettsial Diseases of Man," edited by Rivers and Horsfall.

Dr. Edwin H. Lennette, chief of the laboratory, contributed chapters on serologic reactions in viral and rickettsial infections, and on Q fever. Dr. Harald Johnson prepared a chapter on rabies.

Dr. Johnson is a member of the Rockefeller Foundation staff assigned to the Viral and Rickettsial Disease Laboratory, where he has been working the last four years.

For Your Patients—

A Personal Message to YOU:

As your personal physician I consider it both a privilege and a matter of duty to be available in case of an emergency. Consequently, I thought it would be a good precaution if—on this gummed paper which can be pasted in your telephone book or medicine cabinet—I listed the numbers where I can be reached at all times. They are:

_____ OFFICE

_____ HOME

Sincerely,

_____, M.D.

MESSAGE NO. 2. Attractive, postcard-size leaflets printed on gummed paper, you to fill in telephone numbers and your signature. Available in any quantity, at no charge, as another service to CMA members. Please order by Message Number from CMA, PR Department, 450 Sutter, San Francisco.

WOMAN'S AUXILIARY
TO THE CALIFORNIA MEDICAL ASSOCIATION

WE CAN ASSURE YOU, gentlemen, that our motto for this year . . . "Cooperate and Achieve" . . . has been satisfactorily fulfilled, insofar as was possible in this ten-month year.

Due to the pressure of the advanced date for our annual convention, every county auxiliary accelerated its program in a determined effort to present a report comparable to recent years.

The preliminary records of all of them give promise of a substantial increase in all departments. When the final records are compiled at the close of the fiscal year on the county level, we hope to show the normal expectancy in yearly growth.

In an early issue of CALIFORNIA MEDICINE we will give you the facts and figures to substantiate our advance judgment and we're sure you will be pleased with the scope of our influence.

As a result of the energetic work of our treasurer, Mrs. Charles Sprague, and the state and county membership chairmen, we can report an increase in membership as of this date, and we hope to increase it even more before the May deadline.

We have stressed "community service" particularly, and an effort is being made to poll the number of hours that auxiliary members devote to their individual community voluntary health organizations and civic activities.

It is gratifying to know that nearly all the various women's organizations have more than a fair share of physicians' wives on their rosters—in most instances in leadership capacities. Topping the list in this respect are the P.T.A., the Y.W.C.A., the Girl Scouts and the Cancer Society, followed by A.A.U.W., the Heart Association and many child welfare organizations.

Hundreds of Student Nurse Clubs are directed by members of the auxiliary. Local high schools are most enthusiastic about this program.

The baby sitter groups, "GEMS" (Good Emergency Mother Substitutes) have hundreds of teen-agers enrolled. also directed by auxiliary members.

Nurses' scholarships and loan funds have increased, and annual parties and benefits continue to supply the funds to support the American Medical Education Foundation and Physicians' Benevolence.

We have every reason to be proud of the work of physicians' wives. They are proving to be outstanding leaders. And as members of the Auxiliary to the medical societies, they are of ever increasing value to the profession.

It is our proud privilege to be a part of what you as individuals and as members of your profession represent. Incidentally, we might add that we are seriously aware of the fact that, as your profession goes, so goes our way of life.

Much hard work goes into these activities to promote good will and understanding for the fundamentals of good public health and medical standards as you hope to keep them.

This has been a banner year in the growth of closer relations between the Association and the Auxiliary.

In proportion to your encouragement and help, our enthusiasm rises and our work becomes a pleasure. We appreciate everything you do to help us. Without your cooperation, it wouldn't be possible to accomplish the tremendous results that we achieve.

Your San Francisco office personnel doesn't hesitate when we make a request. Mr. John Hunton and Bob Thomas exert every effort to be of assistance to us, interceding for us with the Council—publishing *The Courier*, which, by the way, continues to be the top medical auxiliary publication in the nation. Doctor Wilbur and Mr. Edwards have been most generous with extra space in CALIFORNIA MEDICINE. Our grateful appreciation to them for their kind remarks, too.

Special consideration by the Council and by our Advisory Board has given us confidence, as well as the assurance that we need, of your appreciation.

The members of our board of directors have concentrated this year on improving organizational policies to strengthen our assistance to the counties.

The stimulation motivated by the unprecedented cooperation of the Association has created the enthusiasm for efficient action by the members of the Auxiliary board of directors, which has undoubtedly reacted on the county level, where the actual working program has achieved such fabulous results.

With your continued interest and support, we will continue to grow.

Thank you for all the courtesies extended to me personally this year.

MRS. NEWELL JONES
President, Woman's Auxiliary to the California Medical Association

NEWS & NOTES

NATIONAL • STATE • COUNTY

LOS ANGELES

Reappointment of **Dr. Joseph M. de los Reyes** of Glendale to the California State Board of Medical Examiners for a four-year term was announced recently by the office of Governor Edmund Brown. Doctor de los Reyes was first appointed in February, 1955.

* * *

The Medical Advisory Board of the **Myasthenia Gravis Foundation** wishes to call attention of the members to the Second International Symposium on Myasthenia Gravis, which will be held in Los Angeles at the Statler Hotel on Saturday and Sunday, April 18 and 19. A wide selection of papers dealing with the anatomy, physiology and biochemistry of muscle and the clinical and pathological features of myasthenia gravis will be presented by many world wide authorities.

* * *

A distinguished achievement award for "significant contributions to human health and welfare" has been made to **Dr. Tracy J. Putnam,** neurologic surgeon associated with the Cedars of Lebanon Hospital. The award was announced by the board of medical editors of *Modern Medicine,* which annually makes the awards.

Doctor Putnam was specifically cited for "A lifetime of fruitful endeavor in teaching and practice and neurologic research into the nature of convulsive disorders."

MERCED

Dr. Shelby M. Hicks of Merced has been appointed to the California State Board of Medical Examiners by Governor Edmund Brown. Dr. Hicks replaces Dr. Clayton Mote of San Francisco, who had served since October, 1947.

SAN DIEGO

The 1959 annual convention of the California chapters of the **American Physical Therapy Association** is to be held April 4 and 5 at the U. S. Grant Hotel in San Diego. On Saturday, April 4, at the general meeting, a distinguished panel of speakers will present an informative program concerning many phases of geriatrics and the physical therapist's approach to the patient.

SAN FRANCISCO

Dr. Robert Stone, professor and head of the department of radiology, University of California School of Medicine, San Francisco, was awarded the gold medal of the American College of Radiology at the annual meeting of the organization in Chicago in February. The medal, highest award by the college, is given for outstanding contribution to radiology.

* * *

Dr. Norman Kretchmer, an associate professor at Cornell Medical School, will succeed Dr. Robert H. Alway as head of Stanford University's department of pediatrics. The appointment, effective July 1, was announced today by Dr. Alway, who now is dean of Stanford Medical School. Dr. Ruth T. Gross, associate professor, has been acting head of pediatrics for the past two years.

Dr. Kretchmer last year received the American Academy of Pediatrics' E. Meade Johnson Award for distinguished pediatric research.

* * *

The **San Francisco Heart Association** has announced the opening of a contest for The William J. and Dorothy Fish Kerr Prize of $350 for the best original observation in the clinical field of cardiovascular disease and embracing diagnosis and treatment, with particular emphasis upon the use of the special senses and the sense of touch at the bedside.

Any intern, resident or resident fellow in any hospital in the San Francisco Bay Region (all the counties which are contiguous with San Francisco Bay) is eligible. The applicant should submit a written description of the observation he has made, and with it a supporting letter from the chief of the hospital service in which the observation was made.

These materials should be submitted before April 15, 1959, to Charles A. Noble, Jr., M.D., chairman, Awards Committee, San Francisco Heart Association, Inc., 259 Geary Street, San Francisco 2.

* * *

The 36th annual meeting of the **American Orthopsychiatric Association** will be held in San Francisco, at the Sheraton-Palace Hotel, March 30 to April 1, 1959.

GENERAL

An **Oregon Cancer Conference** is to be held July 16 and 17, 1959, in Portland under the joint sponsorship of the Oregon State Medical Society, the Oregon Division of the American Cancer Society, the University of Oregon Medical School and the Oregon Academy of General Practice. The conference is planned for midsummer as a special feature of the Oregon Centennial celebration.

A block of rooms has been reserved at the Hotel Multnomah for physicians wishing to attend the conference. A copy of the complete program and hotel reservation forms may be obtained by writing to Roscoe K. Miller, executive secretary, Oregon State Medical Society, 1115 S. W. Taylor Street, Portland 5, Oregon.

* * *

The **American College of Obstetricians and Gynecologists** will hold its annual meeting in Atlantic City, April 6 to 8, with general sessions in Convention Hall. An attendance of about 2,000 physicians is anticipated.

Further information may be had by writing to Mr. Donald F. Richardson, executive secretary, The American College of Obstetricians and Gynecologists, P. O. Box 749, Chicago 90.

* * *

The **American College of Chest Physicians** will hold its silver anniversary meeting at the Ambasador Hotel, Atlantic City, June 3 to 7, 1959. The scientific program will include prominent speakers on all aspects of heart and lung diseases. In addition to formal presentations, there will be a number of symposia, round table luncheon discussions, postgraduate seminars, and motion pictures.

Fireside conferences, inaugurated in 1955, will feature more than 60 experts in chest disease leading discussions on topics of current interest.

* * *

The **Biennial Western Conference on Anesthesiology** will be held in Phoenix, Arizona, April 1 to 4, at the Westward Ho Hotel. Further information may be obtained from Boyden L. Crouch, M.D., 301 West McDowell Road, Phoenix.

POSTGRADUATE
EDUCATION NOTICES

THIS BULLETIN of the dates of postgraduate education programs and the meetings of various medical organizations in California is supplied by the Committee on Postgraduate Activities of the California Medical Association. In order that they may be listed here, please send communications relating to your future medical or surgical programs to: Mrs. Margaret H. Griffith, Director, Postgraduate Activities, California Medical Association, 2975 Wilshire Boulevard, Los Angeles 5.

UNIVERSITY OF CALIFORNIA AT LOS ANGELES

Diagnostic Radiology. Friday and Saturday, March 20 and 21. Twelve hours. Fee: $55.00 (includes lunch).

Multidisciplinary Approach to the Management of Sports Injuries. Wednesday and Thursday, March 25 and 26. Thirteen hours. Fee: $15.00 (includes lunch).

The Place of Adrenal Steroids in the Treatment of Disease. Friday and Saturday, April 3 and 4. Fourteen hours. Fee: $15.00.

Current Developments in Nutrition (Arrowhead). Friday, Saturday and Sunday. April 24, 25 and 26. Ten hours. Fee: $50.00 (includes board and room).

Clinical Laboratory Interpretations. Thursday, Friday and Saturday, May 21, 22 and 23. Nine hours.*

Techniques of Hypnosis. Monday, Tuesday and Wednesday morning, June 15, 16 and 17. Fifteen hours. Fee: $65.00.

Advanced Techniques and Application of Hypnosis. Wednesday afternoon and Thursday and Friday, June 17, 18 and 19. Fifteen hours. Fee: $110.00.

Sterility. Friday and Saturday, July 24 and 25. Twelve hours.*

Anesthesiology. Wednesday, Thursday and Friday, August 5, 6 and 7. Fifteen hours.*

Three Symposia at University of California Residential Conference Center, Lake Arrowhead:

Pediatric Cardiology. Sunday through Wednesday, August 16 through 19. Fifteen hours.* Guest speaker: John Lind, M.D., Stockholm, Sweden.

Emotional Problems in Office Practice. Wednesday through Sunday, August 19 through 23. Fifteen hours.*

Seminars in Internal Medicine. Sunday through Wednesday, August 23 through 26. Fifteen hours.*

Clinical Traineeships—Anesthesia and Dermatology. Dates by arrangement. Minimum period—two weeks. Fee: Two weeks, $150.00; four weeks, $250.00.

Contact: Thomas H. Sternberg, M.D., Assistant Dean for Postgraduate Medical Education, U.C.L.A., Los Angeles 24. BRadshaw 2-8911, Ext. 7114.

*Fees to be announced.

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

Diagnostic Radiology. Friday through Tuesday, March 20 through March 24. Thirty-five hours. Fee: $80.00.

Infection and Immunity (Children's Hospital). Saturday, March 21. Seven hours. Fee: $12.50.

Advances in Occupational Health. Thursday and Friday, March 26 and 27. Fourteen hours.*

Enzymes—Basic and Clinical Aspects. Thursday, Friday and Saturday, April 2, 3 and 4. Twenty-one hours.*

Weekend Seminars for Medical Technologists. Saturday and Sunday, April 4 and 5. Ten hours. Fee: $15.00.

Nursing in Rehabilitation. Monday to Friday, April 27 through May 15. One hundred and five hours. No fee.

Ear-Nose-Throat. Friday and Saturday, May 15 and 16. Fourteen hours. Fee: $40.00.

Pediatrics. Wednesday through Saturday, June 17 through 20. Twenty-eight hours.*

Fundamental Practices of Radioactivity and the Diagnostic and Therapeutic Uses of Radioisotopes. Two or three month course limited to one enrollee per month. Fee: $350.00.

Contact: Seymour M. Farber, M.D., Assistant Dean, Department of Continuing Medical Education, University of California Medical Center, San Francisco 22. MOntrose 4-3600, Ext. 665.

STANFORD UNIVERSITY SCHOOL OF MEDICINE

Morning Clinical Conferences, each Monday, Room 515. *Contact:* D. H. Pischel, M.D., Professor, Division of Ophthalmology, Stanford University School of Medicine, 2398 Sacramento St., San Francisco 15.

Conference on Physiology and Therapeutic Uses of Adrenal Steroids. March 20 and 21, at Stanford Campus in Stanford. No fee.

Postgraduate Conference in Office Dermatology. March 20 and 21. Fee: $50.00.

Symposium on Adrenal Steroids. Friday and Saturday, March 27 and 28. Twelve hours.*

Annual Spring Postgraduate Conference in Ophthalmology. April 6 through 10, at Stanford Eye Bank, San Francisco. Fee: $100.00. *Contact:* Mrs. Alice Crouch, Postgraduate Secretary, Stanford Medical School, 2398 Sacramento St., San Francisco 15.

UNIVERSITY OF SOUTHERN CALIFORNIA, LOS ANGELES

Cardiac Resuscitation. Sponsored by the Los Angeles County Heart Association each Wednesday throughout the year, 4 to 6 p.m. USC Medical Research Building, Room 211, 2025 Zonal Avenue. Residents and interns of Los Angeles County, and all armed forces medical personnel admitted without fee. Tuition for all other physicians $30.00. (Each session all-inclusive.)

Basic Home Course in Electrocardiography. One year postgraduate series, electrocardiogram interpretation by mail. Physicians may register at any time and receive all 52 issues. Fifty-two weeks. Fee: $100.00.

Advance Home Course in Electrocardiography. One year postgraduate series, electrocardiogram interpretation by mail. Fifty-two issues: $85.00. Physicians may register at any time.

Fluid and Electrolyte Balance. March 20 through 22. Hotel Statler, Los Angeles. In addition to introductory lectures, there will be supervised practice on cases which demonstrate electrolyte and water imbalance. Tuition: $75.00.

SPECIAL ANNOUNCEMENT: Last summer a postgraduate refresher course held in Hawaii was so successful that the USC School of Medicine will offer another refresher course in Hawaii and on board the *S.S. Lurline* from July 29 to August 14. (As a time and money saver, round trip air travel is also possible July 29 to August 10.)

Contact: Phil R. Manning, M.D., Associate Dean and Director, Postgraduate Division, University of Southern California School of Medicine, 2025 Zonal Avenue, Los Angeles 33. CApital 5-1511.

COLLEGE OF MEDICAL EVANGELISTS

GENERAL SURGERY AND SURGICAL SPECIALTIES. Full-Time Basic Science Course. Accredited by the American Board of Surgery.

Surgical Anatomy—Head and Neck (14 periods, 63 hours), April 22 through June 3. Tuition: $75.00.

Surgical Anatomy—Head and Neck (12 periods, 24 hours), April 22 through June 3. Tuition: $35.00.

Each Six Months. Anesthesiology (6 months, full-time). Vacancy occurs each six months. Limited to 2 students. Tuition: $350.00.

For information contact: Chairman: Committee on Postgraduate Medicine, College of Medical Evangelists, 1720 Brooklyn Ave., Los Angeles 33.

CALIFORNIA MEDICAL ASSOCIATION POSTGRADUATE COURSES

POSTGRADUATE INSTITUTES

San Joaquin Valley Counties in cooperation with College of Medical Evangelists, March 19 and 20, Hotel California, Fresno. Chairman: Owen Steinbach, M.D., 3004 Fresno St., Fresno.

Southern Counties in cooperation with University of California, San Francisco, April 23 and 24, Disneyland. Chairman: E. F. Cain, M.D., 200 N. Palm, Anaheim.

West Coast Counties in cooperation with Stanford University School of Medicine, May 14 and 15, La Playa Hotel and Golden Bough Theater, Carmel. Chairman: Chester G. Moore, Jr., M.D., 440 E. Romie Lane, Salinas.

North Coast Counties in cooperation with UCLA School of Medicine, June 5 and 6, Hoberg's Ranch, Lake County. Chairman: Lee Zieber, M.D., 1177 Montgomery Dr., Santa Rosa.

Sacramento Valley Counties in cooperation with University of Southern California School of Medicine, June 25 and 26, Tahoe Tavern, Lake Tahoe. Chairman: Robert H. Quillinan, M.D., 616 Alhambra Blvd., Sacramento.

Audio Digest Foundation, a nonprofit subsidiary of the C.M.A., now offers (on a subscription basis) a series of hour-long tape recordings designed to keep the physician abreast of current happenings in his particular field. Composed of practice-useful abstracts from 600 leading journals, with short lectures and editorial comments from prominent physicians, Audio Digest offers programs covering general practice, surgery, internal medicine, obstetrics and gynecology, and pediatrics.

Audio-Digest plans to begin a new series of programs covering the specialty of Anesthesiology. The first of these will be issued early this year. Those wishing to be charter subscribers to this tape-recorded review of what is new and important in the field of Anesthesiology should write to Mr. Claron L. Oakley, Editor, 1919 Wilshire Boulevard, Los Angeles 57, HUbbard 3-3451, for order form and further information.

Contact: Claron L. Oakley, editor, 1919 Wilshire Blvd., Los Angeles 57.

Medical Dates Bulletin

MARCH MEETINGS

Pioneers Memorial Hospital 9th Annual Postgraduate Medical and Surgical Convention. March 20 and 21, program by University of Colorado Medical School. *Contact:* George Jaquith, M.D., secretary, Route 1, Box 70, Brawley, Calif.

Southern California Society of Gastroenterology Meeting, March 24, 8 p.m., at Los Angeles County Medical Association headquarters. *Contact:* Sherman M. Mellinkoff, M.D., secretary, UCLA Medical Center, Los Angeles 24.

Nevada Division, American Cancer Society First Annual Seminar, March 25 and 26, Riverside Hotel, Reno. *Contact:* Ralph Nelson, executive director, P. O. Box 55, Carson City, Nevada.

San Francisco Heart Association Nurses Institute "Care of the Cardiac Patient," March 25 and 26, 8:30 a.m. to 3:30 p.m., Marina Jr. High School, 3500 Fillmore Street, San Francisco. *Contact:* Jean Sullivan, program director, 259 Geary Street, San Francisco.

American Orthopsychiatric Association 36th Annual Meeting, March 30 through April 1, Sheraton-Palace Hotel, San Francisco. *Contact:* Donald Shaskan, M.D., chairman, Arrangements Committee, Veterans Administration, 49 Fourth St., San Francisco 3.

APRIL MEETINGS

American Group Psychotherapy Association 5th Annual Western Regional Meeting. April 2 and 3, Sheraton-Palace Hotel, San Francisco. 9:00 a.m. to 5:00 p.m. each day. *Contact:* H. S. Morgenstern, M.D., Langley-Porter Clinic, University of California Medical Center, San Francisco 22.

California Tuberculosis and Health Association and California Trudeau Society Annual Meeting, April 2 through 4, Villa Motor Hotel, San Mateo. *Contact:* Mrs. Mary C. French, 130 Hayes Street, San Francisco.

AMERICAN ACADEMY OF GENERAL PRACTICE. April 6 through 9, San Francisco. *Contact:* Mr. M. F. Cahal, executive secretary, Volker Blvd. at Brookside, Kansas City 12, Missouri.

LOS ANGELES COUNTY HEART ASSOCIATION Workshop on Work Simplification Techniques for Physicians, Nurses, Occupational Therapists, Physical Therapists, Dietitians, Social Workers. April 7, 1959, Southern California Gas Co., 810 South Flower Street, Los Angeles, 9 a.m. to 4:30 p.m. *Contact:* Rea M. Schneider, M.D., chairman, Heart of the Home Subcommittee, 660 S. Western Avenue, Los Angeles 5.

LONG BEACH SURGICAL SOCIETY "Clinic Day"—Lectures and Round Table Discussions. April 11, 2:00 p.m., Virginia Country Club, 4602 Virginia Road, Long Beach. *Contact:* F. Harriman Jones, M.D., secretary-treasurer, 211 Cherry Ave., Long Beach 2.

AMERICAN ACADEMY OF PEDIATRICS Spring Meeting, April 13 to April 15, Sheraton-Palace Hotel, San Francisco. *Contact:* Charles H. Cutler, M.D., state chairman, Northern California Chapter, 2615 I Street, Sacramento.

AMERICAN ACADEMY OF NEUROLOGY. April 13 through 18, Statler Hotel, Los Angeles. *Contact:* Joseph M. Foley, M.D., Boston City Hospital, Boston, secretary.

CANCER COMMISSION, CALIFORNIA MEDICAL ASSOCIATION Cancer Conference for VENTURA COUNTY MEDICAL SOCIETY. April 14, Oxnard.*

AMERICAN SURGICAL ASSOCIATION. April 15 through 17, Fairmont Hotel, San Francisco. *Contact:* W. A. Altemeier, M.D., secretary, Cincinnati General Hospital, Cincinnati 29.

PALO ALTO MEDICAL CLINIC Second Annual Medical Symposia: Cardiology and Immunology, April 18, 8:30 a.m., Clinic Auditorium. *Contact:* John F. Weigen, M.D., program chairman, Palo Alto Medical Clinic, Palo Alto, Calif.

BAKERSFIELD SURGICAL SOCIETY Meeting, April 18, 8:30 p.m. *Contact:* Chas. P. Marvin, M.D., 2628 G Street, Bakersfield.

AMERICAN SOCIETY OF INTERNAL MEDICINE 3rd Annual Meeting. April 19, Conrad Hilton Hotel, Chicago. *Contact:* Clyde C. Greene, Jr., M.D., assistant secretary-treasurer, 350 Post Street, San Francisco 8.

AMERICAN COLLEGE OF PHYSICIANS Meeting, Conrad Hilton Hotel, Chicago, April 20-24, 1959. *Contact:* Mr. E. R. Loveland, executive secretary, 4200 Pine Street, Philadelphia 4.

HAWAII MEDICAL ASSOCIATION Annual Meeting, April 23 through 25, Hilo. *Contact:* Miss Lee McCaslin, executive secretary, 510 S. Beretania Street, Honolulu 13.

MAY MEETINGS

LOS ANGELES COUNTY HEART ASSOCIATION Annual Membership Meeting, May 6, 12 noon, Statler Hotel. *Contact:* C. A. Alexander, executive director, 660 S. Western Avenue, Los Angeles 5.

THE NEVADA ACADEMY OF GENERAL PRACTICE Annual Meeting, May 21 through 23, Riverside Hotel, Reno. Program by University of Southern California School of Medicine. *Contact:* Roy M. Peters, M.D., chairman, 475 S. Arlington Avenue, Reno, Nevada.

CALIFORNIA HEART ASSOCIATION Annual Meeting, May 22 through May 24, 1959. Scientific Session and Directors Meeting, Lafayette Hotel, Long Beach. *Contact:* J. Keith Thwaites, executive director, 1428 Bush Street, San Francisco 9.

SUMMER AND FALL MEETINGS

WESTERN BRANCH, AMERICAN PUBLIC HEALTH ASSOCIATION Annual Meeting. June 2 through 5, Sheraton-Palace Hotel, San Francisco. *Contact:* Mrs. L. Amy Darter, secretary-treasurer, 2151 Berkeley Way, Berkeley 4.

ROCKY MOUNTAIN CANCER CONFERENCE, Scientific only, July 22 through 23, 9:00 a.m., Brown Palace Hotel, Denver. *Contact:* N. Paul Isbell, M.D., chairman, 835 Republic Building, Denver 2, Colorado.

NEVADA STATE MEDICAL ASSOCIATION, Annual Session, jointly with Reno Surgical Society, August 19 through 22, Mapes Hotel, Reno. *Contact:* Nelson B. Neff, executive secretary, P. O. Box 188, Reno.

SAINT JOHN'S HOSPITAL Postgraduate Assembly, September 10 through 12, Saint John's Hospital, Santa Monica. *Contact:* John C. Eagan, M.D., director, Postgraduate Assembly, 1328 22nd Street, Santa Monica.

WASHINGTON STATE MEDICAL ASSOCIATION Annual Meeting, September 13 through 16, Olympic Hotel, Seattle, Washington. *Contact:* Ralph W. Neill, executive secretary, 1309 Seventh Avenue, Seattle, Washington.

OREGON STATE MEDICAL SOCIETY Annual Meeting, September 23 through 25, Medford, Oregon. *Contact:* Mr. Roscoe K. Miller, executive secretary, 1115 S.W. Taylor St., Portland 5, Oregon.

SAN FRANCISCO HEART ASSOCIATION 29th Annual Postgraduate Symposium on Heart Disease. September 30, October 1 and 2, 9 a.m. to 5 p.m. daily, St. Francis Hotel, San Francisco. *Contact:* Lawrence I. Kramer, Jr., executive director, 259 Geary Street, San Francisco 2. YUkon 2-5753.

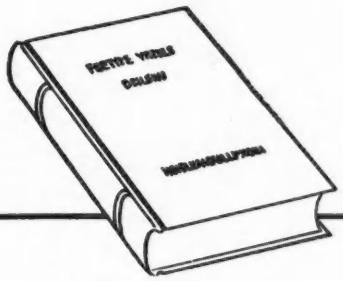
WESTERN INDUSTRIAL MEDICAL ASSOCIATION, INC. 18th Annual Meeting, held in conjunction with Third Western Industrial Health Conference, all day October 2 and 3, Statler Hotel, Los Angeles. *Contact:* A. C. Remington, M.D., medical director, AiResearch Mfg. Co., 9851 Sepulveda Blvd., Los Angeles 45.

CALIFORNIA SOCIETY OF INTERNAL MEDICINE Annual Meeting, October 2 through 4, Miramar Hotel, Santa Barbara. *Contact:* Mrs. Mildred B. Coleman, executive secretary, or Clyde C. Greene, Jr., M.D., secretary-treasurer, 350 Post Street, San Francisco 8.

CALIFORNIA LEAGUE FOR NURSING Annual Meeting, October 8 through October 10, U. S. Grant Hotel, San Diego. *Contact:* Ruth I. Jorgensen, general director, Room 202, 465 Post St., San Francisco 2.

AMERICAN ACADEMY FOR CEREBRAL PALSY Annual Meeting, November 30 through December 2, Statler Hotel, Los Angeles. *Contact:* Margaret H. Jones, M.D., local arrangements chairman, associate professor of pediatrics, UCLA School of Medicine, Los Angeles 24.

*Contact: Walter E. Batchelder, M.D., Medical Director, C.M.A. Cancer Commission, 450 Sutter Street, San Francisco 8.



THE PHYSICIAN'S *Bookshelf*

TREATMENT OF BREAST TUMORS—Robert S. Pollack, M.D., F.A.C.S.; Clinical Instructor in Surgery, Stanford University School of Medicine; Clinical Instructor in Surgery (Oncology), University of California School of Medicine; Assistant Chief of Surgery, Mount Zion Hospital, San Francisco, California; Consulting Surgeon, Oakland Veterans' Administration Hospital; Consulting Surgeon, Oakland Naval Hospital, Oakland, California. Lea & Febiger, Philadelphia, 1958. 147 pages, with 47 plates and 16 text figures, $6.00.

This comprehensive but brief volume is devoted to the treatment of breast tumors including those of cystic mastitis. The classification used is simple and workable and avoids the impractical detail so frequently encountered in such classifications.

Diagnostic criteria are briefly discussed. The technical procedures of excisional biopsy and removal of benign tumors and other pathological processes are described and illustrated.

As would be expected the major portion of the book deals with the various aspects of carcinoma. The importance of exploration of small tumors is stressed. The author's criteria of operability which are reasonably widely accepted are presented. The practical difficulties confronting the surgeon in determining operability are well covered.

Consideration is given to the indications for simple, modified radical, standard radical and extended radical mastectomy. The procedures are described and illustrated.

The chapters on irradiation list the indications for its use and the methods employed are outlined. The limitations of irradiation in cancer of the breast are well discussed.

Additive and subtractive hormone therapy is well handled. The proper use of estrogens, androgens and adrenal cortex hormones is presented. Oophorectomy, adrenalectomy and hypophysectomy are evaluated as adjuncts to therapy.

In controversial areas the author presents opposing views. An effort is made to arrive at fair and balanced conclusions without being dogmatic. The bibliography is extensive and up to date.

The brevity and readability of this work recommend it. The author has succeeded in presenting a surprising amount of material in 142 text pages. It is a good source of ready reference.

JOHN W. CLINE, M.D.

* * *

RORSCHACH TEST DIAGNOSIS—A Textbook in Rorschach Test Diagnosis, for Psychologists, Physicians and Teachers—Ewald Bohm, Ph.D. Translated by Anne G. Beck, M.A., and Samuel J. Beck, Ph.D., Professional Lecturer, University of Chicago, Associate, Michael Reese Hospital. Grune & Stratton, New York, 1958. 322 pages, $7.75.

Since the Second World War there has been increasing concern, both in this country and in other parts of the world, about the one-way character of scientific literature, i.e., the U. S. sends its texts and journals to other countries, but we tend to know little about the material from them. Many more scientists of other lands know English than we know their languages. Increased travel and international meetings are helping in part to increase the communication among scientists and professional people of the many countries, but there still is great need for us to be better informed. Thus it was rewarding to this reviewer to read the Beck translation of Dr. Bohm's text on *Rorschach Test Diagnosis*.

Dr. and Mrs. Beck, prominent in national and international literature on the Rorschach test, have translated from the German volume and made available a view of European use and understanding of the test. The "Translators' Preface" by the Becks surveys the text succinctly and is in itself an admirable review. Bohm's clinical skill and his enthusiasm with the Rorschach test are clearly apparent, together with his knowledge of both European and American literature on the test.

The text is complete in its presentation, beginning with instructions on the method of administering the test, recording and scoring responses, and summarizing them. Also reviewed are intelligence, affectivity, typologies ("Schizaffinic" and "Ixothymic" will be strange to American readers and the latter must translate into his own concepts these descriptions), the neuroses, psychopathies (constitutional or "genuine," and "organic pseudopsychopathies"), depressions, and psychoses. The volume closes with a review of the literature on the test in its use with children, instructions about administration, and cautions about interpreting Rorschach test data from children.

IVAN N. MENSH, Ph.D.

* * *

EMOTIONAL PROBLEMS OF CHILDHOOD—Edited by Samuel Liebman, M.D., Medical Director, North Shore Hospital, Winnetka, Ill.; Clinical Assistant Professor of Psychiatry, University of Illinois College of Medicine. J. B. Lippincott Company, Philadelphia, 1958. 176 pages, $5.00.

There continues to be a search for an adequate book on the emotional problems of young children suitable for a wide variety of practitioners. This collection of articles is an excellent step in this direction. The book covers the psychological aspects of the relations between children and their parents from pregnancy forward. There are special chapters on speech disorders and the handicapped child. Considerable emphasis is given to behavior problems in adolescents and the general management of puberty and sex in adolescents.

On the whole, the articles are well written, clear, and surprisingly even, despite a variety of authors. The two chapters on speech disorders and delinquency do not exactly represent the same school of psychological thinking as do the other papers, and perhaps are not as valuable for this reason. By contrast, the two articles covering parent-child relationships and the psychological development of infants are excellently presented.

This is a recommended book for all those whose practice deals with parents concerned about their children.

HENRY H. WORK, M.D.

CHLOROMYCETIN (Chloramphenicol) — Antibiotics Monographs, No. 8—Theodore E. Woodward, M.D., and Charles L. Wisseman, Jr., M.D., University of Maryland School of Medicine, Baltimore, Maryland; with the collaboration of Harry M. Robinson, Jr., M.D., George Entwisle, M.D., Fred R. McCrumb, Jr., M.D., and Merrill J. Snyder, Ph.D. Foreword by Joseph E. Smadel, M.D. Medical Encyclopedia, Inc., 30 East 60th Street, New York 22, N. Y., 1958. 159 pages, $4.00.

This is the eighth in a series of monographs produced through the efforts of Dr. Henry Welch, director of the Division of Antibiotics of the Food and Drug Administration. Each of these small books is written by a specialist on a particular antibiotic and presents the laboratory and clinical aspects of the drug as a synthesis of the published evidence with the author's personal experience. The present volume on chloramphenicol is undoubtedly one of the best of the series. Drs. Woodward and Wisseman were part of the team assembled by Dr. Smadel in 1948 to study chloramphenicol, the first antimicrobial drug which had a marked therapeutic effect on rickettsial diseases. During their initial studies in Malaya they observed the effect of chloramphenicol in salmonella infections as well as scrub typhus and soon thereafter it became apparent that the drug was likewise valuable in a host of other bacterial infections. With the recognition of the "broad spectrum" nature of chloramphenicol came its large scale abuse: Millions of doses were consumed to treat the "common cold" and as an inevitable consequence the toxic side effects (particularly blood dyscrasias) gained publicity quite out of proportion to their real incidence. As a result only negligible quantities of chloramphenicol were prescribed for several years and the drug did not regain its proper place in the medical armamentarium until 1955-57 when its frequent effectiveness against drug-resistant staphylococci was reemphasized. For some reason the authors fail to describe this dramatic up-and-down in the fame and fortune of chloramphenicol. That is one of the very few faults that can be found with the monograph. It condenses in a concise and factual manner the chemical, pharmacological and clinical properties of the drug as they emerged from 738 references and the author's own large experience. There are particularly good chapters on rickettsial diseases, salmonellosis, bacterial meningitis and staphylococcal infections.

Although the book is probably too detailed for the practicing physician, it might do well as a source of ready reference in his library and I suspect that the volume could be obtained without cost from any sales representative of the manufacturer of chloramphenicol. The monograph will be indispensable for medical libraries.

ERNEST JAWETZ, M.D.

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CLINICAL OBSTETRICS AND GYNECOLOGY—December 1958—A Quarterly Book Series—Volume 1, Number 4—Symposium on Operative Obstetrics, Edited by J. Robert Willson, M.D.; and Symposium on Genital Cancer, Edited by Daniel G. Morton, M.D. Published by Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, 49 East 33rd Street, New York 16, N. Y., 1958. $18.00 per year for four consecutive numbers issued quarterly (by subscription only).

This is another in the series on clinical obstetrics and gynecology published by Paul B. Hoeber, Inc. It contains symposia on operative obstetrics, edited by J. Robert Willson and on genital cancer, edited by Daniel G. Morton. Like the preceding symposia each contains a series of discussions on the important aspects of the respective subjects by authors who have been interested in their particular assignments. This means that we have presented representative contemporary thought. For the most part the chapters are concise and to the point. While I believe that these volumes were aimed at physicians in general practice it seems to me that they have a much wider applicability. The presentations would certainly be of interest to students, residents, and to specialist obstetrician-gynecologists as expressing the present best approved practices.

The symposium on operative obstetrics contains chapters on Dystocia Due to Large or Abnormal Fetus, Breech Extraction, Forceps Delivery, Cesarean Section, Obstetric Version, Sterilization and Therapeutic Abortion and several others. Among the contributors are Thomas R. Goethals, Chas. A. Stevenson, R. A. Cosgrove, Roy G. Holly, Robert Barter and Keith P. Russell. It would be impossible to attempt a description of the contents of each chapter. For the most part the viewpoints expressed are representative, sound and reliable. To single out one point, it does seem to me that Goethals plugs delivery of the breech by means of operative decomposition and extraction at full dilatation more than it deserves. I believe that his view is not really representative of current authoritative opinion.

The symposium on cancer of the female genital tract contains treatises on Early Diagnosis by Herbert Traut, Radiation Treatment of Cervical Cancer by Herbert Schmitz, Operative Treatment of Cervical Cancer by Joe Meigs, Exenteration Operations by Alexander Brunschwig, and Ovarian Cancer by Howard Taylor. Also covered are carcinoma of the vulva and vagina, the endometrium and the Fallopian tubes.

The treatment of these subjects is entirely clinical, of course, and on a down-to-earth practical level.

DANIEL G. MORTON, M.D.

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EMERGENCY TREATMENT AND MANAGEMENT—Second Edition—Thos. Flint, Jr., M.D., Director, Division of Industrial Relations, Permanente Medical Group, Oakland and Richmond, California, and Chief, Emergency Department, Permanente Medical Group, Kaiser Foundation Hospital, Richmond, California. W. B. Saunders Company, Philadelphia, 1958. 539 pages, $8.00.

In this 539-page book the author has placed in outline form conditions encountered in the emergency departments of clinics and hospitals that require immediate management. From the foreword one reaches the conclusion that this source of material is particularly adaptable in urgent and life saving situations. The book itself does not fulfill that objective.

For example, under acute abdominal conditions there are listed thirty-six different illnesses including porphyria, vitamin deficiency and radiculitis. Such a complex list of illnesses could not serve a useful purpose to an experienced physician and would add to the confusion of an inexperienced intern.

This book includes an outline on pruritis ani and insomnia which seems out of place in a text on emergency room treatment and management.

On the complimentary side, the book has an up-to-date section on the diagnosis and management of cardiac arrest, head injuries, drowning, bullet wounds, and such accidents encountered under dramatic circumstances in an emergency ward or hospital.

This book would serve satisfactorily for the senior medical student or intern on an emergency service. It would be a readily available source in outline form to help more systematically organize his thinking in emergency states.

Twenty-two pages are used to outline "Administrative, Clerical and Medicolegal Principles and Procedures."

This book could well be recommended for the graduating medical student or intern anticipating service on the emergency ward or as a ready reference in acute emergencies when time would not permit him to read more extensively.

JOHN G. WALSH, M.D.